

INFANTILE PARALYSIS
IN VERMONT



University of
Connecticut

LIBRARIES

GIFT OF

Health Sciences Library
at
Hartford Hospital

**INFANTILE PARALYSIS
IN VERMONT
1894-1922**

PRINTED BY
THE VERMONT PRINTING COMPANY
BRATTLEBORO, VERMONT

INFANTILE PARALYSIS IN VERMONT

1894-1922

A Memorial to
CHARLES S. CAVERLY, M. D.



BURLINGTON, VERMONT
STATE DEPARTMENT OF PUBLIC HEALTH
1924

COPYRIGHT 1924
BY STATE DEPARTMENT OF PUBLIC HEALTH
BURLINGTON, VERMONT

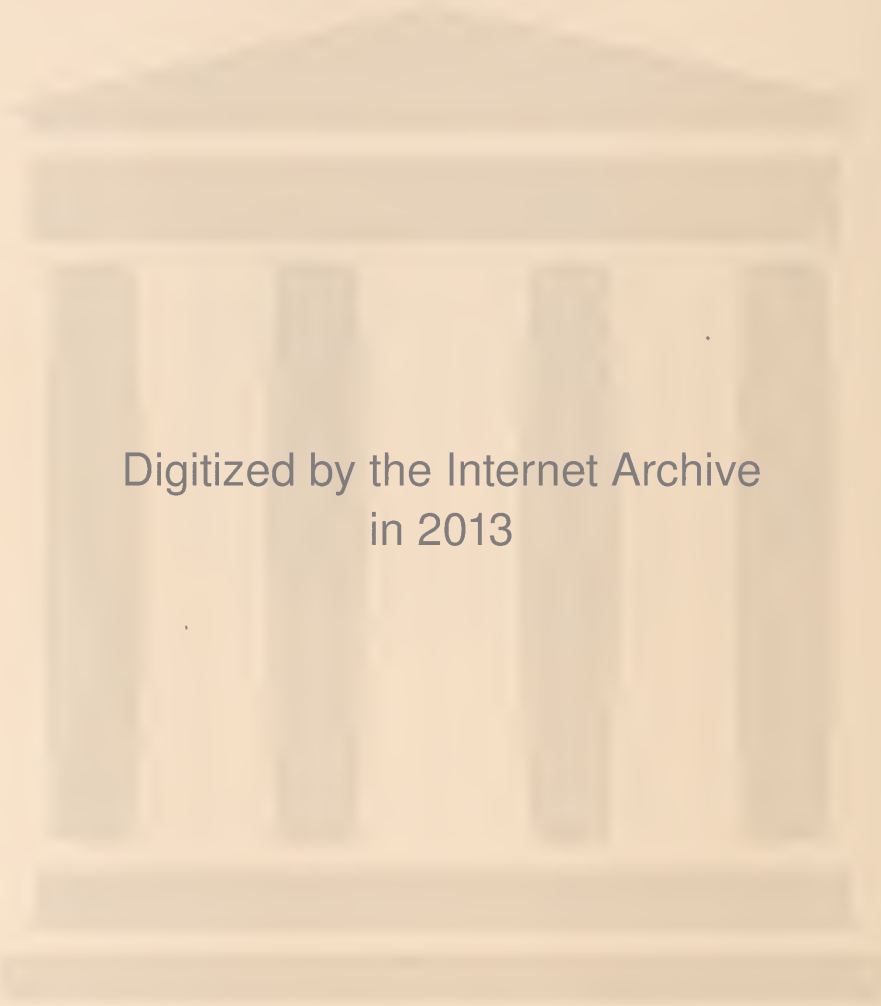
PRINTED IN THE UNITED STATES OF AMERICA

F 411.51

In 3

PREFACE

THIS volume has been prepared as a memorial to Dr. Charles S. Caverly. It consists, in the main, of his writings on the subject of poliomyelitis in Vermont, to which are added certain contributions from the men who worked with him or after him, and a statement of the present status of the work instituted by him.



Digitized by the Internet Archive
in 2013

<http://archive.org/details/infantileparalys00cave>

CONTENTS

PREFACE	5
INTRODUCTION	9
DR. CHARLES S. CAVERLY, A BIOGRAPHICAL NOTE	13
CONTRIBUTIONS OF CHARLES S. CAVERLY, M.D.,	
Preliminary Report of an Epidemic of Paralytic Disease, Occurring in Vermont, in the Summer of 1894	15
Notes of an Epidemic of Acute Anterior Polio- myelitis	21
Anterior Poliomyelitis in Vermont in the Year 1910	39
Anterior Poliomyelitis in Vermont in 1911	56
Epidemic Poliomyelitis in Vermont in 1912	69
Epidemic Poliomyelitis, A Review of the Epi- demics of 1914 and 1915	97
Infantile Paralysis (Poliomyelitis) in Vermont, 1916-1917	144
CONTRIBUTIONS OF THE AFTER-CARE DEPARTMENT,	
The Treatment of Infantile Paralysis—Prelimi- nary Report Based on a Study of the Vermont Epidemic of 1914	201
<i>By Robert W. Lovett, M.D.</i>	
Infantile Paralysis in Vermont—A Report of Progress of Cases between January, 1915, and July, 1915	232
<i>By Robert W. Lovett, M.D., and E. G. Martin, Ph. D.</i>	
Certain Aspects of Infantile Paralysis	240
<i>By Robert W. Lovett, M.D., and E. G. Martin, Ph. D.</i>	
The Spring Balance Muscle Test	252
<i>By Robert W. Lovett, M.D., and E. G. Martin, Ph. D.</i>	

A Plan of Treatment in Infantile Paralysis	263
<i>By Robert W. Lovett, M.D.</i>	

History of the After-Care of Poliomyelitis in Vermont	275
<i>By Robert W. Lovett, M.D.</i>	

Organization, Aim, and Plan of Work of the After-Care Department for Poliomyelitis in Vermont	302
<i>By Bertha E. Weisbrod, R.N.</i>	

CONTRIBUTIONS OF THE RESEARCH DEPARTMENT,

A History of the Work in Poliomyelitis of the Research Laboratory, Vermont State Board of Health	307
<i>By W. L. Aycock, M.D.</i>	

The Care and Treatment of Acute Poliomyelitis in Vermont	312
<i>By W. L. Aycock, M.D.</i>	

Neutralization of the Virus of Poliomyelitis by Nasal Washings	318
<i>By Harold L. Amoss, M.D., and Edward Taylor, M.D.</i>	

Carriage of the Virus of Poliomyelitis, with Subsequent Development of the Infection	337
<i>By Harold L. Amoss, M.D., and Edward Taylor, M.D.</i>	

The Treatment of Acute Poliomyelitis—Preliminary Note on Use of Hypertonic Salt Solution and Convalescent Human Serum	348
<i>By W. L. Aycock, M.D., and Harold L. Amoss, M.D.</i>	

Experiments on Local Specific Therapy in Poliomyelitis—The Utilization of Hypertonic Solutions in the Serum Treatment of Experimental Poliomyelitis	355
<i>By W. L. Aycock, M.D., and Harold L. Amoss, M.D.</i>	

INTRODUCTION

THE question as to why man alone of all the animals became civilized is answered by one historian in this wise: "All animals gain a certain wisdom with age and experience, but the experience of one ape does not profit another. Learning among animals below man is individual, not coöperative and cumulative." This may or may not be the answer, but it is certainly true that today men have a feeling of what might almost be called moral obligation to pass on to others the things that they have found out for themselves.

Although men may battle with men, and nations with nations, man knows neither personal grudge nor national enmity in the fight against a common foe that threatens all mankind, such as disease. In the field of medicine and public health man has conquered and will continue to conquer only by cooperative effort and the benefit of cumulative experience. The knowledge of the cause of a disease and its treatment must not die with him who discovered it; and when a disease is still unconquered it is quite as important that all the ground gained should be held, and all possible information as to the enemy's strength and weakness should be known to those who are still fighting the battle.

Infantile paralysis, or poliomyelitis, still baffles science as to its cause and prevention; and while not a new disease, having been fairly well described by Michael Underwood as early as 1774, it has occurred in epidemic form only in comparatively recent years. The first known epidemic of any considerable size in the United States occurred in Vermont in the summer of 1894. This was reported by Dr. Charles S. Caverly, President of the State Board of Health.

Not only was Vermont to be associated with the history of infantile paralysis because of this first epidemic, but

later epidemics made heavy inroads upon the comparatively small population of the state, leaving many crippled children to be cared for. This situation became so serious that after an epidemic of 306 cases in 1914, through the efforts of Dr. Caverly and the generosity of an anonymous friend of the state whose interest he enlisted, a special fund was provided for the study of the cause and treatment of infantile paralysis.

Thus it happened that from being the first state in which the disease occurred in epidemic form, Vermont also became the first to undertake on a state-wide scale the after-care of the victims of infantile paralysis. The special fund, as stated, also provided for research as to the cause and transmission of the disease, and in this field, too, much valuable knowledge has resulted from the efforts of those carrying on the work.

The history of infantile paralysis in Vermont really falls into three divisions: (1) the epidemiologic study of the disease; (2) the treatment of affected cases; and (3) the work of the research department. It is the belief of those who have been connected with the work that the data collected along these lines form a contribution to the knowledge of infantile paralysis and are worthy of permanent record. They are therefore compiled in this present form. In the pages that follow will be found Dr. Caverly's collected reports on the epidemiology of the disease; certain articles by Dr. R. W. Lovett and those working with him on the plan of state-wide treatment adopted, and the results of this treatment; and the data obtained from the laboratory studies conducted by the research department under the supervision of Dr. Simon Flexner and carried on by Dr. H. L. Amoss, Dr. Edward Taylor and Dr. W. L. Aycock.

No piece of constructive work of any magnitude was ever accomplished by one man alone, but it is usually true that long before the first step is taken the idea has assumed defi-

nite form in the mind of one person, who is the leader and inspiration of the other workers. He has seen the need and has caught the vision of the goal to be achieved. Perhaps, as the plans grow and the work develops, it may go beyond the fondest dream of him who gave it its first impulse, or it may never reach the goal which he foresaw. Dr. Caverly did not live to see the disease against which he had fought so long and so untiringly really conquered and the fear of future epidemics removed; but he did live to see many of the victims of infantile paralysis in Vermont well on the road to happy, useful, and in some cases normal lives, and to know that real contributions to the knowledge of the disease had been made by those working in cooperation with the State Board of Health under his presidency.

The fight against infantile paralysis is by no means ended, and the disease still presents to the medical profession a most perplexing problem. In so far as this volume may be of use to those engaged in its solution, it is hoped that it may be worthy of the memory of him to whom it is inscribed—Dr. Charles S. Caverly, a leader and tireless worker in the field of the treatment and prevention of infantile paralysis, President of the Vermont State Board of Health from 1891 until the time of his death in September, 1918.

DR. CHARLES S. CAVERLY

DR. CHARLES SOLOMON CAVERLY was born in Troy, New Hampshire, September 30, 1856, and came of an old New England family, his great great-grandfather, Philip Caverly, having served as a soldier in the Revolutionary War. He was educated in the high schools of Pittsford and Brandon, and prepared for college at Kimball Union Academy, in Meriden, New Hampshire. In 1878 he graduated from Dartmouth College, where he was a member of the Phi Beta Kappa; and after receiving the degree of A.B. there he went to the University of Vermont, from which he received the degree of M.D. in 1881. Here he was class leader and the recipient of three prizes. Subsequently he studied for eighteen months at the College of Physicians and Surgeons in New York, and began practice in Rutland, Vermont, in 1883, being associated with Dr. Middleton Goldsmith.

Although always successful in private practice, his interest in public health movements became evident at an early date. He became a member of the Vermont State Board of Health in 1890, and its President and animating spirit in 1891, which office he held until the time of his death. He was also Professor of Hygiene and Preventive Medicine at the University of Vermont, from which he received the honorary degree of Sc.D. on account of his distinguished service in the interest of public health. He died October 16, 1918, after a brief period of influenza with complications.

Early in his work Dr. Caverly became interested in poliomyelitis, and in December, 1894, he published in the *New York Medical Record* an article describing the epidemic occurring in Vermont in the summer of that year. This report stood out as the best contribution, in America at least, to our knowledge of the disease up to that time. He recognized

the abortive cases, which were not so named and recognized until a great many years later, and he gave a very intelligent and advanced account of the behavior of the affection. He never lost his interest in the condition, and his biennial reports on infantile paralysis in Vermont have been of great value to the medical profession.

In 1914, when an epidemic of 306 cases occurred in the state, he set seriously to work to see what could be done toward helping our knowledge of the disease. Vermont is a small state with comparatively few inhabitants, and 306 cases represented a very high incidence. In this year and in subsequent years he gave a great amount of time to the direction of the work for the care and prevention of poliomyelitis, in which he was intensely interested and with which he was actively associated up to the time of his death.

Dr. Caverly was also very deeply interested in the cure and prevention of tuberculosis, and his help in the establishment and work of the Pittsford Sanatorium, and later the Preventorium at Essex, was very great.

I knew him first in 1914, when I went at intervals to Vermont to conduct the treatment of the cases of infantile paralysis there, and I was greatly impressed by his personality and his public spirit. We were thrown much together, and I became intimately acquainted with him, an intimacy which continued up to the time of his death. He was a quiet, cultivated man, with a singleness of purpose which was striking, a lack of pretense which was always charming, and an ability which was perfectly evident. He was universally respected and looked up to for his attainments, his accomplishments, and his personal character. He kept himself in touch with the progress of medicine in all departments; his manner of conducting matters of business was always quiet, efficient, and unassuming; and when he died he left behind him the feeling that a man of real ability, high personal character, and great usefulness had been taken away.

ROBERT W. LOVETT.

PRELIMINARY REPORT OF AN EPIDEMIC OF
PARALYTIC DISEASE, OCCURRING
IN VERMONT, IN THE
SUMMER OF 1894*

By CHARLES S. CAVERLY, M.D.

EARLY in the summer just passed, physicians in certain parts of Rutland County, Vermont, noticed that an acute nervous disease, which was almost invariably attended with some paralysis, was epidemic. The first cases observed occurred in the city of Rutland and the town of Wallingford, appearing about the middle of June. The disease prevailed chiefly in the city of Rutland up to about the middle of July, when other towns about this city began to report cases.

From my own observation and conversation with other physicians, and the general feeling of uneasiness that was perceptible among the people in regard to the "new disease" that was affecting the children, I determined during the last of July to undertake a systematic investigation of the outbreak, in my capacity as a member of the State Board of Health. I sought from all the physicians practicing in the area which I knew to be affected by the disease such information in regard to their own cases as it was possible for them to contribute. This investigation, undertaken in an official capacity, soon convinced me that this region had been affected by an epidemic of nervous disease very rarely observed.

For the reason that this outbreak is of such an unusual character and especially interesting to the general practitioner everywhere, I am sure that I shall be pardoned in using the facts which I have succeeded in collecting for the general benefit of the profession, as well as for the public.

*From the Yale Medical Journal, November, 1894.

It is yet too early to make a complete or exhaustive report of this epidemic. I will merely, in what I shall have to say in this article, briefly summarize the statistics which I have so far secured, and refer only incidentally to the exact nature of the disease.

The cases, of which I have collected reports, more or less complete, number 123. They date from June 17th to September 1st. The territory covered is mainly the narrowest part of the Otter Creek Valley in Rutland County, bounded on the east by the Green Mountain range and on the west by the Taconic range, having no natural boundaries north and south. This valley is approximately fifteen miles wide, including the sides of the bounding mountains, and thirty miles long.

Of the 123 cases of which I have notes, all but six occurred in this valley. Through this valley flows the Otter Creek from south to north, a sluggish stream, which during the present summer is said to have been lower than ever before. The population is about 26,000, of which probably 18,000 live in the manufacturing and quarrying centers of Rutland, Proctor and West Rutland. The towns which have been most affected by this epidemic are those immediately on the Otter Creek. I can best illustrate the character of the epidemic by citing a few cases of which I have reports.

Case I was a boy three years old, American; previous health good; very active child; stronger than his brother a year or two older; no apparent cause; taken with moderate fever; very irritable; tongue coated; an apparent attack of indigestion, though no diarrhoea or nausea. After two or three days, the febrile symptoms abated, and his parents called the attention of the physician to his inability to use his legs. The extensor muscles of the thigh seemed to be chiefly affected. He could not walk; could not stand steadily for ten days. At the end of that time, he began to use his legs in walking by holding on to chairs, and in three weeks' time had fully recovered their use.

Case II was a boy, three and one-half years old, Irish, sturdy child, one of three or four children. The only apparent cause, playing too hard on a hot day. Had fever, 102° to 104° , for two days; incontinence of urine for ten days. On the third day paralysis of both legs. Loss of patellar reflex; considerable hyperæsthesia. After the initial symptoms had subsided, there was only slight reaction in the muscles of both lower extremities to faradism. After two weeks the right leg improved rapidly, the left not so fast. After six weeks he was able to take hold of chairs and walk a little. The paralysis and wasting, after ten weeks, was confined to the left gluteal and lower spinal muscles and has resulted in spinal curvature.

Case XCIII was a boy, two years old, American; first had an attack of indigestion, from which he apparently recovered, when he was taken for a long carriage drive and at once developed fever with erythematous rash over the body; some muscular rigidity of the neck and back, and, after a few days, paralysis in both legs with loss of patellar reflex. After two months has not improved much.

Case CXIV was a boy, six years old; previous health good; apparent cause, chilling the body when heated; had high fever, temperature 104° ; vomiting; acute symptoms lasting six days. On the sixth day had paralysis of the right arm, followed on the seventh by paralysis of the left leg. The extensors of the left thigh and right deltoid muscles are now paralyzed (after ten weeks) and somewhat wasted.

Case IV was a boy, six years old; was taken with convulsions while playing in the street, convulsions lasting nine hours; moderate fever, rapid pulse; vomiting; muscular rigidity of the neck and back; hyperæsthesia of extremities; very restless, no paralysis noted; death on the sixth day.

These cases are fair illustrations of those encountered through this epidemic. The cases have not been confined to children. I find one case reported in a man seventy years

old, who exhibited almost the same train of symptoms as a child in a mild form of this disease. He lost the use of both legs for seven days and after that rapidly recovered their use.

I find also several cases among persons from fifteen to forty years of age, and several deaths. The ages of those reported were as follows: Under six years, eighty-four; six to fourteen years, twenty; over fourteen years, twelve. Stated as "between a few months and nine years," seven.

I find that the sex of those under six years of age, where stated, was exactly twice as often males as females. Over fourteen years of age, the same ratio prevailed. Of those between six and fourteen whose ages are given, nine were males and two females. The number of those stated to have had some form of paralysis is 110. Of this number fifty have fully recovered at the time of my report, ten had died, leaving fifty who were apparently permanently disabled. The exact location of the initial paralysis when it occurred, and that still left at the time my reports are made, I have not as yet had time to tabulate. Of the 123 cases of which I have reports, seven died very early and before any paralysis was noted, six are definitely stated to have had no paralysis, one of which died.

It is noted that there have been several degrees of severity in the disease which has prevailed here, or else there have prevailed several diseases. Case I, whose history I have given, represents fairly the mildest cases. Cases II, XCIII and CXIV represent the cases that appear now to be permanently disabled, while Case IV is a fair sample of those which have died.

It will readily occur to physicians that the symptoms noted in these cases are suggestive of two diseases, Cerebro-Spinal Meningitis and Acute Anterior Poliomyelitis. The initial fever, followed in a few days by definite motor paralysis, of which a certain percentage recover in a few weeks,

the rest suffering permanent impairment of some muscles, offers a fair picture of the average case of Poliomyelitis Anterior, while the high fever, muscular rigidity, and hyperæsthesia, are not characteristic of this disease.

The season of the year, the absence of special sense symptoms, especially deafness, as a sequella in this epidemic, the low mortality, the absence of the very characteristic purpuric eruption, are strong arguments against the theory of Cerebro-Spinal Meningitis. It is now well established that the other disease, Poliomyelitis, is occasionally epidemic. Such epidemics have been noted in at least three instances in Europe, and one is reported by Putnam as occurring near Boston. The Stockholm epidemic reported by Medin is in many respects quite similar to the one which I have reported.

Without going further into the matter of diagnosis, as I do not as yet feel warranted in doing, I may say that Dr. A. Jacobi, of New York, whose opinion I am permitted to quote, pronounces this epidemic one of Cerebro-Spinal Meningitis. I should state that Dr. Jacobi's knowledge of our epidemic has been gained solely from my written description to him. Dr. M. Allen Starr, who has taken much interest in this epidemic and has seen ten cases in the chronic stage, very kindly allows me to quote him as of the opinion that the features of our epidemic resemble more nearly Poliomyelitis than Cerebro-Spinal Meningitis. Dr. Charles L. Dana, whose knowledge of this epidemic is likewise gained from what I have written him in regard to it, and from the partial examination of the brain and cord of a fowl which had paralytic symptoms and was taken from a flock in this section that was dying of symptoms suspicious of a nervous disease, says as follows:

"I can most positively state my opinion to be that your cases were mostly cases of Anterior Poliomyelitis." It may be remarked that the microbic or infectious nature of the

disease is generally believed in by us, whose opportunities for observing it have been merely clinical. Further investigation on the brain and cord of the lower animals we hope may throw some light on this point. There is no evidence of its contagiousness, since it has affected almost invariably but a single member of a household.

I might state further that there have been many deaths among horses, attended with symptoms of paralysis, and in which at least one veterinarian tells me he found meningitis. There have been, too, some deaths with similar symptoms among dogs and fowls.

This report is the merest outline of this epidemic, but as I secure fuller facts and have time, I hope to record a complete history of this interesting outbreak. The 123 cases, with eighteen fatalities, probably represent eighty-five or ninety per cent of all that have occurred. It was utterly impossible to secure in any of the fatal cases an autopsy.

For the data on which these observations are founded, I am under obligations to the profession generally throughout this region, who have responded with uniform promptness to my request for facts.

Rutland, Vt., Oct., 1894.

NOTES OF AN EPIDEMIC OF ACUTE ANTERIOR POLIOMYELITIS*

By CHARLES S. CAVERLY, M.D.

THE following "Notes" are the result of an investigation undertaken by me in an official capacity at the time of the outbreak, and since continued through private and professional intercourse.

The epidemic was one of an acute nervous disease whose chief distinguishing characteristic was motor paralysis, more or less complete, of one or more members or groups of muscles, and which prevailed in the State of Vermont, chiefly in a single valley, during the summer of 1894. The results of my investigations, as far as completed at the time, were published in the *Yale Medical Journal* for Nov., 1894, and in the *New York Medical Record* for Dec. 1, 1894. At the time of making these reports, it did not seem possible to speak of the epidemic more definitely than as one of "acute nervous disease of unusual type." A further careful study of the complex features of the epidemic, however, and of the subsequent history of many of the cases, together with the corroborative opinions of many able medical men, seems to clear up any doubt that at the time existed as to the correct diagnosis of the essential disease that prevailed.

I may state at once that I am indebted to Professors A. Jacobi, M. Allen Starr and Chas. L. Dana for very valuable aid in arriving at a diagnosis in this series of cases, as well as for notes of the latest pathologic views of poliomyelitis and a literary résumé of that disease. I am also indebted to my fellow practitioners of Vermont who have kindly placed at my disposal the results of their clinical observations of the epidemic. This paper is necessarily largely a recapitula-

*Read in the Section on Neurology and Medical Jurisprudence at the 46th Annual Meeting of the American Medical Association, at Baltimore, Maryland, May 7-10, 1895.
Reprinted by permission from J. of A. M. A., 1896, XXVI, 1.

tion of the facts heretofore published about this epidemic, with a careful revision of the statistics of the outbreak made from recent observations.

The epidemic, as I have indicated, invaded our valley in the early summer of 1894. It prevailed with increasing severity during July, apparently reached its climax about the first of August, and steadily declined until about the first of October, the last case occurring early in that month.

The early summer was popularly considered unusually hot and dry, though the official figures do not substantiate the former opinion. That it was an exceptionally dry season is manifest from the figures of the United States Weather Bureau of the State, which show that the rainfall during the five months from April to August, inclusive, for this year was one-third less than the average for these months since the opening of the station in the State. The temperature and humidity statistics show little variation from the average. The territory mainly covered by this outbreak is a portion of the Otter Creek Valley, about thirty miles long and from twelve to fifteen wide, including the sides of the bounding mountains. On the east of this part of the valley is the main Green Mountain range, and on the west the Taconic range, which is a northern extension of the Berkshire Mountains of Massachusetts. Otter Creek, the largest stream of water in the State, has its source in the mountains to the south of the affected area, and sluggishly flows in a northerly direction through it, emptying some miles below into Lake Champlain. That part of its course through the affected district is the most populous and likewise the narrowest part of the valley. The city of Rutland is the commercial and geographical center of this area. The towns affected have a combined population of 26,000, of which fully two-thirds dwell in the quarrying and manufacturing centers of Rutland, West Rutland and Proctor.

The starting point of the epidemic, and most of the earlier

cases, were at Rutland. In this city occurred 55 of the 132 cases of which I have notes; 27 of the remainder occurred in the town of Proctor, one-sixth the population of Rutland. This town suffered the worst of any in the valley. The remaining fifty cases were scattered over the rural districts in fourteen towns. The most of these cases occurred at considerable elevation above the creek, and many well up on the Green Mountains. Four of these towns with eight of the cases are not in the Otter Creek Valley. The natural drainage of the valley is the creek, and this stream, below Rutland, carries a large amount of sewage. If the disease had shown any preference for those houses immediately on the stream below Rutland, it might at once be inferred that the low water in a sewerage-contaminated stream had some bearing on the etiology of the disease. But such did not appear to have been the case, except possibly, in the town of Proctor, which is six miles below Rutland and is built on the abrupt bluffs above the stream. Drainage defect in general did not seem to influence the distribution of the disease. The water supply was excluded as an etiologic factor, it being largely from wells in the rural communities, and in the villages from mountain streams and springs. That the general sanitary surroundings and methods of living were in anywise responsible for the outbreak is also more than doubtful, since the disease showed no partiality to that class of the population whose habits and surroundings are the most unsanitary. The so-called laboring classes were oftenest affected, but not out of proportion to their numbers. These classes here, whether among the farming population or in the mills and quarries, have usually pure air, food and water. Hence, general sanitary conditions did not seem to have any influence on the epidemic.

The geologic formation of the valley is not peculiar. The prevailing formation is limestone, and in the range that skirts the western border of the valley is found the chief

marble deposit of Vermont. The valley as a whole is an old lake basin and is pronounced by Prof. G. W. Perry, the State Geologist, as a very ordinary valley.

The outbreak of which I speak consisted of upward of 130 cases of disease in which the commonest clinical manifestation was some degree of motor paralysis of widely varying extent. It will not surprise any one that so large a number of cases presenting a bewildering variety of initiatory constitutional symptoms, as well as local paralyzes, should have proved a very knotty problem for the diagnostician. It was long a question whether this was an epidemic of one, two, or more diseases, and along the established lines of symptomatology and pathology there was no solution of the problem. I have been able to collect histories more or less complete of 132 cases directly affected in this epidemic, and this number probably represents at least 90 per cent of the whole number.

Case 1. Boy, 3 years, American. Hygienic surroundings good; previous health good; active child; stronger than his brother two years older. No apparent cause. Fell sick June 20. Moderate fever, coated tongue, loss of appetite, sluggish bowels. His condition was confidently ascribed to indigestion, and after two or three days the continuance of the symptoms, though in decreasing severity, proved troublesome of explanation. On the third day his parents insisted that he could not use his legs. It was soon evident that this was the case. His reflexes were normal, sphincters unaffected, no anesthesia or noticeable hyperesthesia. The weakness was most marked in the large extensors of the thigh. After the entire subsidence of his febrile symptoms, his muscular weakness began to improve, at first very slowly. In three weeks he had gone on to full recovery.

This case is an excellent illustration of the mildest type of the disease, a type that included about forty cases.

Case 2. Boy, 3½ years, Irish. Hygienic surroundings

fair; sturdy child; most active of a family of three children. Only apparent cause playing too hard on a hot day. Taken with high fever, temperature 102° to 104° F., nausea, general restlessness and headache. Had incontinence of urine, no albuminuria. On third day acute symptoms subsided except the incontinence of urine. It was then noticed that he had lost the use of his legs. Patellar reflexes diminished and considerable hyperesthesia of the legs. There was also diminution of faradic irritability. The left leg improved rapidly, the right slowly. After six weeks was able to stand and take a few steps by taking hold of chairs. After three months the paralysis and wasting were confined to the right glutei and lower spinal muscles. His efforts to walk have brought on a slight spinal curvature. The incontinence of urine continued in this child until Feb. 1, 1895, when it was relieved by circumcision. The paralysis, however, persists in the glutei and lower spinal muscles and promises to be permanent.

This case illustrates a very common phase of this epidemic, and in most of these cases there is probably some permanent impairment of certain muscles.

Case 88. Practice of Dr. Gale, Rutland. Girl, 6 years, American. Previous health had been frail. Had had a spinal curvature since she began to walk. Taken suddenly with high fever, nausea, head- and backache. On the fourth day of the attack she was paralyzed in all the extremities and one side of the face. Febrile symptoms subsided at this time. There was extreme hyperesthesia of the whole body and obstinate constipation from seeming lack of power in the abdominal muscles. Facial paralysis speedily passed off. Hyperesthesia and pains in the joints required the use of morphine for several weeks. After nine months she is still paralyzed in all the extremities, being able to flex the fingers and toes slightly and raise the head. The hyperesthesia has passed off.

Case 116. Practice of Dr. Swift, Pittsford. Boy, 4 years, Italian. Taken with headache, drowsiness and slow hobbling pulse. Little fever. After four days developed strabismus. Improved speedily and at the end of four days was apparently well. Three days later, after playing too hard, had a return of the original symptoms. Headache, drowsiness, no fever, pulse 45. In two days from this time had a convulsion and speedily died.

Case 32. Practice of Dr. Marshall, Wallingford. Woman 21 years. Married and one child of 16 months. Apparent cause fatigue from nursing sick child. First had head- and backache. Pulse 80, temperature 98.6° . On third day pulse 100, temperature 103.5° . Some opisthotonos; bowels regular; urine, 2 pints in twenty-four hours. No albumin, no sugar. Urticarial blotches on the body. During the next three or four days temperature ranged from 100.5° to 102° , pulse about 100. Was unable to speak or swallow. Answered questions by moving the head; in no pain. Sixth day temperature 98.6° ; pulse 60. Remained in this condition five days. On the eleventh day complained of severe pain in the stomach, and neck became rigid; pulse 100, temperature 98.6° . During the next two days pulse became very irregular. Complained of severe pain in the right side of the head and right eye. Died at the end of the second week.

Case 4. Practice of Dr. Fox, Rutland. Boy, 6 years, previous health fair. On two or three occasions had convulsions, presumably due to gastro-intestinal disturbance. Was seized with convulsions while playing on the street; they continued for nine hours. Moderate fever, rapid pulse, vomiting and rigidity of muscles of the neck and back. No paralysis noted during conscious intervals. Retention of urine during the last three days of illness. Death on the sixth day.

These four cases represent various types of the severe form of the disease; among these cases there were eighteen deaths.

There were a great many cases exhibiting rare and interesting phenomena, a detailed report of which would consume too much time. One of these, during an attack of broncho-pneumonia, had loss of speech for two weeks, and paralysis of one arm which recovered in five weeks. One developed paralysis of both legs in connection with pneumonia. The paralysis in one case was confined to the external rectus of one eye. Several, after apparently recovering from the acute symptoms, were again attacked more severely than at first. Two cases, in which the legs were paralyzed, had a concurrent fever with the characteristic typhoid curve. One case was that of a boy of 6 years who had been at the seashore during the summer, and returning to the town of Proctor after the epidemic was apparently on the wane, on September 5, was attacked with the typical symptoms of poliomyelitis on September 30, and is left with impairment of the extensors of one thigh and the glutei. This case is interesting as showing possibly the length of the incubation period of the poison if we class the disease among the infectious disorders.

Without detailing further individual cases, a condensation of my notes presents the following clinical picture of the epidemic:

Age and Sex.—Ninety cases were under 6 years of age; 39 were boys and 22 girls; sex of the remainder not stated. Fifteen cases were between the ages of 6 and 14; 5 were males and 6 females; sex not stated in 4 cases. Fifteen cases were over 14 years; 9 were males and 6 females. In one series of cases, 7 in number, the age is stated as between "a few months and 9 years," and the sex of none is given. In another of 5 cases, neither age nor sex is stated. It is interesting to note in this connection that there were 9 cases in adults upward of 21 years of age. One of these was a man of 70 who had the familiar symptoms of the milder type of these cases with paralysis of both legs, which passed off in

ten days. The other 8 cases were in persons from 21 to 38 years of age. These figures as to age and sex do not differ from those usually given for poliomyelitis. While it is chiefly a disease of childhood it is not exclusively so. Males are vastly more liable to it.

Nationality.—In those cases in which the nationality is stated 41 were American, 17 Irish, 6 French, and one each was Hebrew, Italian and Swede. I know of no significance to attach to these figures.

Previous Health.—Of the 46 cases in which the previous health of the sufferers is given, in 35 it is given as good, and in 11 as poor. It is quite certain that the strong, healthy children preponderated.

Immediate Cause.—The immediate apparent cause is stated in 37 instances. Of these overheating is mentioned 24 times, chilling of the body 4 times, trauma 4 times, while fatigue, typhoid fever, pneumonia and whooping cough are mentioned. There was a general absence of infectious disease as an etiologic factor in this epidemic. The element of contagium does not enter into the etiology either. I find but a single instance in which more than one member of a family had the disease, and as it usually occurred in families of more than one child, and as no efforts were made at isolation, it is very certain that it was non-contagious.

Initial Symptoms—Fever. In most of the cases there was a perceptible rise in temperature at the start, though a few are said to have had none. Of the 56 cases in which the temperature is noted, 27 had a temperature at some time of 103° or more, while in 26 it ranged from 99° to 103°. Three are said to have had “no fever.” The duration of the initial fever, where given, varied from a few hours to two weeks. The four cases, however, that are said to have had a fever for more than a week, probably suffered from some complication or some intercurrent disease. Twenty-six cases had a febrile stage lasting from four to seven days, 7 lasting

from three to four days, 6 lasting two or three days, 2 lasting one to two days, and four for one day or less.

Digestive Organs.—Nausea was a very common symptom and is mentioned as occurring twenty-six times. It was often the first symptom noted and was probably one of the commonest. Gastralgia occurred in few cases. Thirteen cases were said to have had obstinate constipation, and six had a diarrhea.

Urinary Organs.—Two cases had incontinence of urine and in ten cases there was retention. In no case is albuminuria mentioned.

Skin.—Thirty cases are said to have had a simple erythema, and two had urticaria. There was an entire absence of herpetic and purpuric eruptions.

Nervous System.—Convulsions occurred in 12 cases, all under 14 years of age. Muscular rigidity of the neck or back muscles, or both, is said to have occurred in 20 cases, of which 5 were fatal. It is a very significant fact that 36 cases are noted as having hyperesthesia of the skin. Only one is said to have had any anesthesia of the paralyzed member. In several instances soreness of the joints of the affected limb was a very painful symptom. Nine cases are said to have suffered from headache alone, 2 from pain in the back and 23 from both head- and backache. These symptoms were probably commoner than the figures indicate. There was no general tendency to impairment of the special senses. Two cases are said to have had double vision, 3 strabismus, one was blind and one deaf.

Initial Paralysis.—The paralysis which was the leading and most common characteristic of this series of cases, occurred in 119 instances. Of the remaining 13, 7 died before paralysis had time to develop, or it could not be determined whether there was really paralysis or not, and the remaining 6 that had no paralysis, all had a group of symptoms very common in the initial stage in those which were para-

lyzed, such as headache, fever, convulsions or nausea, one or all. In those cases in which the exact day of the paralysis is noted, it is stated to have occurred four times on the first day, eight times on the second, ten times on the third, five times on the fourth, three times on the fifth, once on the sixth, four times on the seventh, and once on the tenth day of illness. It is quite likely that the actual duration of the premonitory symptoms prior to the appearance of the paralysis was often overestimated, since loss of power in the extremities, especially in children, might easily go unnoticed for some time, unless the physician or friends were looking for it. In several instances the loss of power in the legs was the first symptom noticed. The initial paralysis was located as follows:

Both legs	69	cases
Arm and leg, same side	10	"
One arm	5	"
One leg	7	"
Both legs and one arm	4	"
Tongue and throat	2	"
Both arms	3	"
All the extremities	4	"
Extensors of one thigh	2	"
"Variously in the arms and legs"	8	"
External rectus of one eye	1	"
One side of the face	1	"
One arm and the opposite leg	1	"
All the extremities and abdominal muscles	2	"
Stated to have had no paralysis	6	"
Not determined	7	"

Of the six cases that are said to have had "no paralysis" all had distinct nervous symptoms explainable only on the supposition that they belonged to this epidemic. All the seven cases in which it was not certain whether they were paralyzed, died early, often with convulsions, and their occurrence at this time seems to warrant their being included in this series.

Of those cases that are known to have fully recovered according to the latest information I can obtain—

Both legs were paralyzed in	43	cases
Arm and leg, on same side, in	4	"
One arm in	1	"
One arm and both legs in	1	"
External rectus of one eye in	1	"
One leg in	1	"
There was no paralysis in	5	"

That there have been more complete recoveries than this, viz., 56, is quite certain, but I have not been able to trace them.

Fatal Cases.—Eighteen deaths occurred as follows: 1. Boy, 10 years; died within twenty-four hours with convulsions. 2. Boy, 6 years; died on sixth day with convulsions. 3. Boy, 10 months; died on sixth day, paralyzed in both legs. 4. Boy, 4 months; died on sixth day, all the extremities paralyzed. 5. Girl, 11 years; died on third day, no paralysis noted. 6. Girl, 11½ years; died on sixth day, no paralysis noted. 7. Female, 21 years; died on thirteenth day, no fixed paralysis. 8. Male, 19 years; died on fifth day, both legs paralyzed. 9. Sex and age not stated; had paraplegia. 10. Male, 21 years; died on third day, all extremities paralyzed. 11. Sex and age not stated; died with convulsions. 12. Sex and age not stated; had hemiplegia. 13. Girl, 7 years; died on seventh day, all extremities paralyzed. 14. Boy, under 1 year; no paralysis noted. 15. Boy, 4 years; died on second day of relapse, no paralysis of the extremities, but strabismus. 16. Male, 22 years; died on third day, both legs paralyzed. 17. Male, 38 years; died on sixth day, both legs paralyzed. 18. Girl, 11½ years; died on fourth day.

It will be seen that 10 deaths were among males and 5 among females, and that the sex is not stated in three cases. Seven of those that died are known to have been under 6 years, three between 6 and 14 years, while one died at 19 years, two at 21 years, one at 22 years, and one at 38 years. The percentage of deaths among adults is seen to have been very high.

A further analysis of the deaths shows that five of the

cases were paralyzed in the legs, three in all the extremities, and one was hemiplegic. I might state that in the great majority of fatal cases the diagnosis was meningitis. Such a diagnosis was usually not at all inconsistent with the clinical features of the disease. Deducting from the whole number of cases, those which are known to have terminated fatally, and in recovery (74 in all), there remain 58 cases to be accounted for. Just how many of these are still and probably permanently paralyzed, I am not able to state. It was a common experience for a part of the initial paralysis to clear up within the first month, leaving a single member or a single group of muscles weak and wasted. Thus, many cases that at first seemed to be paralyzed in both legs soon improved as to one, and the permanent lesion was seen to be in the other limb or in a few of its muscles. The extensor muscles of the thigh, the glutei, ileopsoas, calf muscles, and anterior tibial group, in the lower extremity, and the deltoid and extensors of the forearm, were frequent sufferers.

Permanent Paralysis.—Of the 58 cases which my report left unaccounted for, I have been able to get reports of 30 which are still maimed, from six to nine months after the initial attack. Of these 16 are stated to be males, and 12 females. Eighteen are under 6 years, 7 are between 6 and 14 years, and 5 are over 14 years of age. Here again we see the high percentage among the older patients. Of these 30 cases—

All the extremities are paralyzed in	1 case
Both arms in	1 "
Extensors of one thigh in	6 "
Glutei and lower spinal muscles in	1 "
Both legs in	6 "
Extensors of one thigh and one leg in	2 "
One leg in	6 "
Glutei alone of one side in	1 "
One foot and ankle in	1 "
Extensors of one hand in	1 "
Both legs, thigh and hips in	1 "
One arm in	2 "
Complete hemiplegia in	1 "

The muscular atrophy in most of these cases is marked though combatted by the usual treatment of rubbing, massage and electricity.

During this epidemic and in the same geographical area, an acute nervous disease, paralytic in its nature, affected domestic animals. Horses, dogs and fowls died with these symptoms.

The only reliable facts which I am able to give of the pathologic conditions in these cases among the lower animals are from the examinations of the cord of a horse that died paralyzed in the hind legs, and from that of the cord and brain of a fowl which was paralyzed in its legs and wings. Dr. W. W. Townsend, of Rutland, who made the examination of the horse, says that the examination of a section of the lumbar portion of the cord showed a "granular degeneration and pigmentation of the ganglion cells of the anterior cornua, and atrophy of the anterior nerve roots." He further states that there was no meningitis in this case. Dr. Charles L. Dana, who made the examination of the fowl, with the aid of Dr. Dunham of the Carnegie Laboratory, found "an acute poliomyelitis of the lumbar portion of the cord and no meningitis." A bacteriologic examination of the same cord by Dr. Dunham gave negative results, and it was found that the inoculating needle did not strike the diseased parts.

It was not infrequently remarked by physicians practicing in this valley at the time of this epidemic, that the usual diseases of children were accompanied with exaggerated nervous symptoms. Headache, convulsions and delirium were common.

It is recorded by Medin, in an epidemic of poliomyelitis which he reports, and to which I shall presently refer, that polyneuritis prevailed with poliomyelitis. The pain, hyperesthesia, and tenderness of the extremities suggest such an explanation in some of our cases. Gowers is also cited by

Putnam (*American Journal of the Medical Sciences*, March, 1895), as speaking of the combined occurrence of poliomyelitis and neuritis. It will be readily seen that it would be quite impossible to reconcile the widely varying phenomena of this epidemic with the established characteristics of any one disease. So it is not strange that local observers differed in their diagnoses. In collecting notes of this epidemic I did not seek any disease by name, endeavoring only to secure histories of such cases as had had well-marked symptoms of acute nervous disease, the paralysis usually being the test symptom. Meningitis, poliomyelitis and neuritis were mentioned with varying frequency, and it is only a careful study of the epidemic as a whole, giving due weight to the predominating symptoms, the paralysis, of course, being the most striking, together with a knowledge of the latest pathology of these diseases, that seems to warrant the conclusion that the essential disease was poliomyelitis. Some of the commonest symptoms seen in our epidemic were entirely foreign to this disease as long described. Likewise, too, its epidemic character and the simultaneous affection of the lower animals. I am especially indebted to Dr. M. Allen Starr for notes of the latest views of the pathology of poliomyelitis.

In *Zeitschrift für Klin. Med.*, 1892, Goldschreider's views, founded on such cases as ended in autopsies, are given as follows: "The disease begins with a very intense congestion of the central arteries of the spinal cord which come up on each side of the central canal and spread out in the gray matter of the posterior horns, but the posterior horns are chiefly supplied with blood from the peripheral arteries, and hence, are less affected when the inflammation is limited to the distribution of these central arteries. After the engorgement of all the arterial twigs, diapedis occurs and the surrounding nervous tissue is permeated by small cells and by serum. It is this choking of the gray matter by the

inflammatory products which leads to the suspension of functional activity, and when, as in many cases, from impoverished nutrition the cells of the anterior horns are actually disintegrated by the inflammatory products, permanent destruction of the nerve tissue ensues. Goldschreider believes, therefore, that the primary condition is a congestion in the domain of a definite set of arteries, quite comparable to the condition occurring in the lung in pneumonia and in the intestine in typhoid fever.

Siemerling, in an article on the "Pathology of Infantile Paralysis" in *Archiv für Psychiatrie*, January, 1894, says: "after a careful review of all the literature we reach, therefore, the following conclusion, that in the pathogenesis of infantile paralysis the inflammatory lesion of the interstitial tissue in connection with a distension of the blood-vessels, especially in the region of the anterior spinal arteries, plays the chief role. A primary inflammation of the ganglion cells in the sense given by Charcot is not to be admitted."

In view of this newer pathology, showing as it does that the initial lesion is not confined to the anterior horns, but that there is a simultaneous invasion of other portions of the cord, the exceptional symptoms seen in our epidemic are rendered explainable.

The results of autopsies made by Rissler were even more striking. He found an acute parenchymatous inflammation of the anterior horns in the cord with degeneration of the ganglion cells and secondary degeneration of the nerve fibers in the association tracts, in the anterior columns and in the anterior nerve roots; also in the nuclei of the hypoglossus, vagus, facial and abducens nerves. In most of the cases the anterior horns were particularly affected; in other cases, also the peripheral nerves and brain cortex. It was possible that all the nerve nuclei in the medulla and pons should be affected. In the light of these recent pathologic

researches taken in connection with the most noted clinical features of this outbreak, viz., the season of the year, the preponderance of cases among children, the widely varying and almost universal paralysis and the low mortality, the conclusion seems unavoidable that the essential disease was poliomyelitis. Under this classification the epidemic at once assumes great importance in several particulars:

1. From the simple fact that it was an epidemic of poliomyelitis.
2. From the great number of cases occurring.
3. From the simultaneous affectation of the lower animals.

While epidemics of poliomyelitis are not unknown or unrecorded, recent authorities speak only vaguely of their occurrence. It has not thus far found a definite place in the descriptions of this disease. The fact that poliomyelitis may occur epidemically, suggests, of course, an infectious origin, a view of the nature of the disease which has only been recently discussed.

I have been able to find the following reference to outbreaks of poliomyelitis of epidemic character, very largely through the assistance of Prof. Jacobi.

In the transactions of the Tenth International Congress (Berlin, 1891), Vol. ii, Prof. O. Medin reports that Bergenholtz in Sweden observed and reported thirteen cases occurring near Lyons, France, four of which were fatal. Medin also cites Eichhorst, who speaks of observations sometimes made of several cases occurring in the same neighborhood. G. Lotmer (*American Journal of the Medical Sciences*, 1843) was told by the parents of a child treated by him for poliomyelitis (teething paralysis), that in a place where they previously lived eight or ten cases had been known within three or four months. Medin's epidemic, however, is the most extensive of any of which I find a record,

and bears in many respects a close resemblance to the Vermont epidemic. The disease appeared in Stockholm in the month of May, 1887, and by August 9 had assumed an epidemic character. Medin saw twenty-nine cases between the latter date and September 23, and knew of forty-four during the summer from May to November. Three of his cases were fatal in the acute state. "There were noticed paralysis of the abducens in five cases, disturbance of speech in a few; in one case paralysis of the tongue, in several of the accessorial; in others, symptoms referable to the vagus. Disturbance of the voice and paralysis of the muscles of mastication and vasomotor paralysis were seen in two fatal cases. In one ophthalmoplegia externa. At the time of the Stockholm epidemic, during August and September, polineuritis appeared and was thought to be due to the same cause. This neuritis was followed by considerable tenderness. These neuritis cases, however, showed no wasting."

It will thus be seen that Medin's observations are very similar to those here recorded, and together with the latest pathologic views of the disease, will seem to mark the neuritis of which he speaks as a varying manifestation of poliomyelitis.

Putnam (*Boston Medical and Surgical Journal*, Vol. cxxix, p. 509) speaks of the unusual prevalence of poliomyelitis in and about Boston during the latter part of the summer of 1893. Twenty-six cases had come to his notice during that season.

That a disease occasionally prevails epidemically suggests a specific poison, a definite toxin, and this phase of the etiology of poliomyelitis has recently received attention from foreign observers as well as from Dana, Putnam and others in this country. Thus far, however, there does not seem to have been any substantial progress made toward isolating any specific microorganism peculiar to this disease.

Our epidemic with that of Medin suggests, though on

purely clinical grounds, the possibility of such a cause. The unfortunate absence of an autopsy in our cases, though strenuous efforts were several times made to secure them, prevents us from throwing any light on this part of the subject. That domestic animals suffered with human beings in our epidemic is a noteworthy fact and one, so far as I can learn, hitherto unobserved. That such was the case cannot be doubted. It has long been known that animals were often attacked by meningitis during an outbreak of that disease in epidemic form. Poliomyelitis has been produced artificially in rabbits and guinea-pigs, but so far I have been unable to find an instance of its spontaneous occurrence simultaneously with the disease in man. This fact again emphasizes the possible infectious character of the disease and lends additional interest to the epidemic here recorded.

ANTERIOR POLIOMYELITIS IN VERMONT IN THE YEAR 1910*

BY CHARLES S. CAVERLY, M.D.

DURING the summer of 1894 an outbreak of poliomyelitis occurred in the Otter Valley in this state which was at the time unprecedented in the number of cases.

This early Vermont outbreak attracts attention by reason of the number of cases involved, also because of certain other features hitherto unnoticed in this disease. These were the facts that lower animals seemed to be affected, that there were *fatal cases* in the human family and that the disease was *not confined to childhood*.

All these phases of anterior poliomyelitis have since received much attention, as epidemics of the disease have recurred with increasing frequency. From 1894 to the present time this disease has increased with alarming rapidity—not only in this country but abroad. Many large epidemics have occurred and the death rate has apparently increased with the epidemic prevalence of the disease.

As would be expected of a disease that is so obviously spreading and recurring in epidemic form, poliomyelitis is being studied now the world over. The features of these epidemics, so suggestive of an infectious cause, are the subject of careful observation in the field and experimental research in the laboratory. Thereby a wholly new literature is rapidly being written into medicine.

With the possible exception of tuberculosis, no disease is now exciting as much interest in medical circles as poliomyelitis.

Two or three decades ago the disease received scant

*Reprinted from *Bulletin of the Vermont State Board of Health*, Vol. XII, No. 2, Dec. 1, 1911.

notice in the text books. The cases were rare—"sporadic"—and usually connected with the early years of childhood, the first dentition. Fatalities were hardly known. The after effects were appreciated as bad, but, fortunately, the rarity of the disease made these less noticeable.

The recent intense study of poliomyelitis has not been without results and promises soon to solve the mysteries surrounding its causation and spread.

The infectious nature of the disease had been pretty conclusively shown by the work of Landsteiner and Popper, as well as by Flexner and Lewis. Emulsions of diseased cords have been used to transmit the disease in a series of monkeys.

It has been shown that the virus persists in the nasal mucosa for a long time. This fact suggests a possible connection with the infective principle of epidemic meningitis. The two diseases have other features in common.

These experiments have also made it possible to diagnose abortive cases, and have shown that urotropin has an inhibiting influence on the appearance of the paralysis. They have shown that the fly may harbor the virus for a period of at least forty-eight hours.

Aside from the results of experimental research in poliomyelitis, the clinical study of epidemics has suggested quite strongly its contagiousness, directly from person to person and by way of third persons.

Aside from the experimental work referred to, the disease has been the subject of considerable study by the Health Departments of Massachusetts, Pennsylvania and Minnesota in this country and by scientists of several European countries. Health officials everywhere are collecting much clinical data, which must eventually prove useful in this connection.

In the collection of the data in regard to the outbreak in Vermont in 1910, and the preparation of the charts and

tables given herewith, the author of this paper has had valuable assistance from Dr. B. D. Adams, inspector of the State Board of Health.

The line of enquiry followed has been quite largely suggested by the published Reports of the Massachusetts State Board of Health on the recent outbreaks in that state.

Since the Vermont epidemic of 1894 this state has not suffered from this disease in anything approaching epidemic form until 1910. In 1910 we had in the state sixty-nine* cases, of which accurate records can be secured. The actual number of cases was greater than this. Many physicians and some health officers were slow to learn that the disease was reportable. Hence some cases escaped official notice.

The history of this outbreak in Vermont is interesting especially because of its possible relation to outbreaks in Massachusetts and the Province of Quebec.

The state of Massachusetts suffered a rather extensive outbreak in 1910. One of the chief foci of the disease in that state was in the city of Springfield. In that city there were 130 cases.† The disease in Springfield began in May, increased to July, when the epidemic culminated, and decreased gradually through August, September and October.

An outbreak of the disease also occurred in the Province of Quebec.

Reliable and complete data in regard to this outbreak are not available, but that there were a considerable number of cases during the summer, there is no doubt.

The affected area in the Province of Quebec was in the neighborhood of Sherbrooke. The outbreak seemed to center about a summer resort of about 500 permanent population, called North Hatley. This resort is between Newport and Sherbrooke. It is on Massawippi Lake and has about 1500 summer visitors, largely from this country. There is direct

*Three additional cases have come to light since the above was written, and too late to tabulate in this Report.

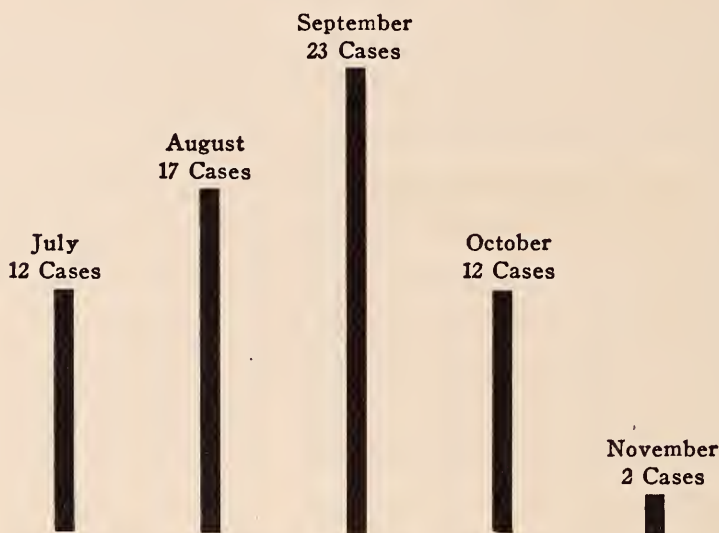
†Annual Report of the Health Department, Springfield, 1910.

railroad communication between Orleans County in Vermont and the places affected on the other side of the border.

The epidemic in this area began in June and increased until August and September. There were only four or five cases reported in Sherbrooke, but the resident doctor at North Hatley estimates that there were twelve cases in that resort that sustained permanent paralysis, besides a considerable number which recovered.

The accompanying chart shows the monthly distribution of cases in our outbreak.

POLIOMYELITIS. VERMONT, 1910. SEASONAL DISTRIBUTION.
VERMONT STATE BOARD OF HEALTH.



The only comment that seems necessary at this point is the fact that the disease began in Vermont two months later than in the Connecticut Valley in Massachusetts. The Canadian outbreak seems to have begun in June, but most of the cases occurred in August and September, coincident with the Orleans County group in Vermont.

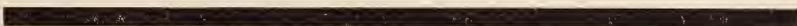

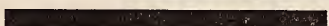
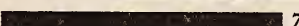

The distribution of the disease in the state is still more suggestive in connection with our neighbors north and south. The accompanying chart shows graphically the relative number of cases by counties and also on the east and

west of the Green Mountains. It will be noted that fifty-one of the sixty-nine cases reported were on the east side of the state. A glance at the map shows the close connection that exists between this side of the state and the affected areas in both Canada and Massachusetts.


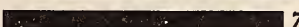



POLIOMYELITIS. VERMONT, 1910.

DISTRIBUTION BY COUNTIES EAST AND WEST.

EAST

Orleans		20
Washington		12
Windham		8
Windsor		7
Caledonia		4
Essex	None	
Orange	None	
Total		51

WEST

Rutland		8
Chittenden		7
Addison		1
Bennington		1
Franklin		1
Grand Isle	None	
Lamoille	None	
Total		18

Proportionate to the population, by far the largest number of cases occurred in Orleans County. After this follows Windham, Washington and Windsor.

POLIOMYELITIS. VERMONT, 1910.

DISTRIBUTION BY COUNTIES IN PROPORTION TO POPULATION.

Orleans	1 to 1,150
Washington	1 to 3,500
Windham	1 to 3,400
Windsor	1 to 4,800
Caledonia	1 to 6,700
Rutland	1 to 6,000
Chittenden	1 to 6,100
Addison	1 to 20,000
Bennington	1 to 21,000
Franklin	1 to 29,000



Distribution of Poliomyelitis in Vermont 1910

Orleans County at the north end of the state is in close connection with the territory affected with the disease in Canada; and Windham, particularly, is almost as closely connected with Springfield in Massachusetts. As to the traceable connection by means of cases between our state and our neighbors, mention will be made later.

The map here shown gives a graphic picture of the geographical distribution of the Vermont outbreak in 1910.

The following charts show the division of the cases by sex and age.

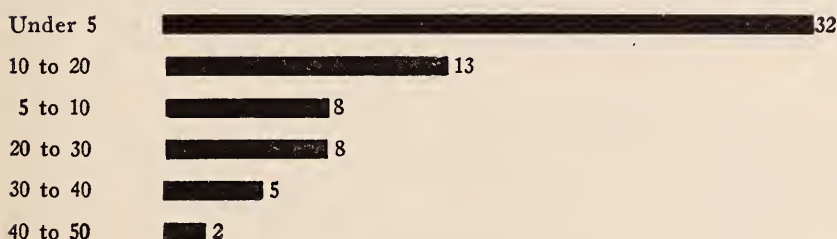
POLIOMYELITIS. VERMONT, 1910.

SEX



POLIOMYELITIS. VERMONT, 1910.

DIVISION OF CASES ACCORDING TO AGE.



An interesting fact developed by the chart for age is that a little over 20 per cent of the cases were over twenty years of age. In this connection it should be stated that the average age of all cases was ten years and six months. The average age of all those who had some degree of permanent paralysis was a little under ten years. The average age of those who died was thirteen years and four months; and of those that fully recovered nineteen years and ten months.

The nationalities affected were:

American	47
French Canadian	6
Irish	3
English	2
Polish	2
Russian	2
Not stated	7

In regard to the surroundings of the patient, the following statistics have been secured:

Age of House

Under ten years	10
Over ten years	56
Not stated	3

Detached House or Tenement House

Detached house	43
Tenement house	15
Not stated	11

Soil About the House

Dry....43	Wet....26	Not stated....2
-----------	-----------	-----------------

Surroundings of the Patient's House

Steam railway within fifty rods	14
Street railway on the street	5
Stream or pond within fifty rods	37
Manufacturing establishment within fifty rods	10

Domestic Animals Kept

Reported as keeping cats, dogs, hens, cows, horses and pigs:			
42 kept cats.		12 kept cows.	
31 kept dogs.		13 kept hens.	
12 kept horses.		7 kept pigs.	

Proximity of Animal Quarters

Animal quarters within 20 feet of house	16
Animal quarters at greater distance	28
Not stated	25

An effort was made to secure reliable figures as to the presence of parasites or vermin in the houses affected. The observation of the inspector and the answers to questions which he was able to secure were so obviously unreliable that the figures are considered worthless. It may, however,

in a general way be stated that the disease was quite apt to occur in houses that obviously harbored vermin of various sorts.

Water Supply

The water supply of infected houses was usually good. The exact figures are as follows:

Wells	6
Private aqueduct	1
Public supply	11
Stated as good	23
Stated as fair	7
Spring water	21

Sewer Facilities

The sewer facilities were probably on an average poor. They are stated as:

"Dry closet"	44
Connected with the public sewer	23
Not stated	2

Cases Located in Railroad Towns

Number of cases that occurred in a town located on a railroad ..	58
Number of cases that occurred in towns off the railroad	11

Family History

Good (no case of chronic disease, like tuberculosis, cancer, diabetes, Bright's disease, or rheumatism in immediate family)	53
Fair (one case of such disease only in immediate family)	11
Bad (two or more such cases in immediate family)	5

Health a Month Prior to Attack

Gastro-intestinal disorder	6
Coryza	2
Bronchitis	1
Whooping cough	2
Tonsillitis	1
Not well—no definite disease	5
Not stated	1
Good	51

In this connection it is proper to give the incidence of other diseases in the same town:

In towns where whooping cough prevailed there were	8 cases.
In towns where the grippe prevailed there were	3 cases.
In towns where mumps prevailed there were	4 cases.
In towns where scarlet fever prevailed there were	4 cases.
In towns where paratyphoid prevailed there were	9 cases.
In towns where diphtheria prevailed there were	2 cases.
In towns where typhoid fever prevailed there were	4 cases.
In towns where chicken pox prevailed there was	1 case.

In regard to school attendance, our outbreak began and reached its crisis during the summer vacation. *Only four* cases are reported as having attended school within one month.

Seventeen cases had been away from home within the month. Of these, *nine* had been in towns or cities where the disease was present, although it was not known in any of these cases that the person had been in contact with the disease. *Eight* cases gave a history of having been in direct contact with other cases. Two others came in contact indirectly. *Five* others had possibly been in contact directly or indirectly.

One patient, who died, twenty-seven years old, from Brattleboro, visited in Springfield, Mass., within a fortnight of his attack. This case occurred September 20, rather late in the epidemic.

The earliest date of any of the Brattleboro group of cases that have been secured was July 16. This was a case in Brookline that had not been out of town within a month.

Another case in Irasburg, twenty-two years old, attended the fair in Sherbrooke, Canada, two weeks before attack. This case, also fatal, occurred September 16, also rather late in the epidemic.

Of the early cases in the Orleans County group, one small child was brought from Topsfield, Mass., about thirty days before illness began. No other early case can be traced to another state.

The obvious possibility of mild unrecognized "abortive" cases, occurring in connection with the outbreak, makes any attempt to trace connection between centers of infection very difficult and, in the present state of our knowledge of the etiology of the disease, almost entirely speculative.

While none of the earlier cases in Vermont are directly traceable to visits to neighboring centers of infection, the coincidence of the outbreaks in time and the direct railroad

communication with these other centers must still remain suggestive of a common source.

There were three instances of two cases in the same family. In one other instance a case occurred in the same family ten years ago. In still another instance, the case was a nurse, who was caring for a case of poliomyelitis.

Ten cases had been in the water within a week of the attack.

Six had been exposed to extreme heat.

Five had been chilled.

Six patients gave a history of recent injury—usually a fall with blow on the head.

First Symptoms Noted

Fever	69 cases
Pain	45 cases
Tenderness	42 cases
Vomiting	38 cases
Diarrhœa	10 cases
Headache	19 cases
Head cold	14 cases
Delirium	10 cases
Chills	14 cases
Sore throat	9 cases

Symptoms During Attack

Disturbance of digestion occurred in	63 cases
Bladder symptoms occurred in	22 cases
These symptoms were retention	17 cases
Incontinence	4 cases
Frequent urination	1 case

Pain and tenderness was a prominent symptom, occurring in sixty cases, at some time during the attack.

The pain was general in	14 cases
The pain was in the extremities in	28 cases
The pain was in neck and back in	18 cases

The accompanying chart shows the incidence of the paralysis.

POLIOMYELITIS. VERMONT, 1910.

DAY OF DISEASE PARALYSIS OCCURRED.

1 to 3		46
4 to 7		11
After 7		4
Not stated		8

It should be stated that the paralysis appeared simultaneously with the febrile symptoms or was the first symptom noted in *eight cases*.

The distribution of the paralysis is shown by the following chart:

POLIOMYELITIS. VERMONT, 1910.

DISTRIBUTION OF PARALYSIS IN THE INDIVIDUAL.



The condition of the cases six to nine months after the attack is shown by the following chart:

POLIOMYELITIS. VERMONT, 1910.

CONDITION OF CASES SIX TO NINE MONTHS AFTER ATTACK AND DEATHS.



The death rate of poliomyelitis has been variously stated by late investigators at from 8 to 15 per cent.

The death rate among our cases in 1910 by age periods was as follows:

- Of 32 cases under five years, 4 died, or 12+ %.
- Of 8 cases between 5 and 10 years, 0 died, or 0%.
- Of 13 cases between 12 and 20 years, 3 died, or 23+ %.
- Of 8 cases between 20 and 30 years, 2 died, or 25%.
- Of 5 cases between 30 and 40, 0 died, or 0%.
- Of 2 cases between 40 and 50, 1 died, or 50%.

While the mortality in our outbreak was 14.49 per cent among all ages, the mortality among those under ten years was 10 per cent. Among those over ten years, it was 21.42 per cent. This mortality is rather higher than in some recent reports. These figures would probably be reduced if we had been able to secure fuller reports of the mild or abortive cases in this outbreak. The mortality figures, however, corroborate the findings of other observers, viz., that the death rate is higher in older subjects.

The investigation of the foregoing outbreak of poliomyelitis revealed two rather significant instances of paralytic disease in the lower animals.

One of these instances was the occurrence of a form of paralytic disease in calves. In the language of the owner, an exceptionally observing and intelligent man, he had a herd of eighteen calves, pastured on an interval meadow about September 1. They were provided with good water. There were no swampy places and no sewage reached the river, which flowed through the meadow. These calves were seven months old. The average weight was about three hundred pounds.

About the first of September, one was noticed not to take his feed and it soon became paralyzed in the hind legs and in less than twenty-four hours died. When the carcass was being drawn away, the other calves gathered about. There was some frothy substance expelled from the nostrils of the dead calf. The owner noticed that one of the other calves put its nose to the nose of the dead calf "in a smelling way"

and near enough to get some of the froth upon its own nose, whereupon he remarked that if this is a contagious disease, he was fearful the other calf might acquire it. This other calf was marked for identification. In less than twenty-four hours that calf was paralyzed behind the same as the other and very soon it died. In this manner five calves, one after another, of this herd, died— all within ten days. No veterinary saw them.

The farmer made what he terms “an unprofessional examination” of the carcass of the second calf that died, and states that there was no discoloration of the tongue, neck, legs or lungs. He thought the lungs appeared to be normal and he found only about two teaspoonfuls of liquid in the chest cavity. The pleura was not inflamed and the bowels appeared to be normal.

I quote this layman’s description of the disease in his herd, as stated, because it is that of a disinterested and rather accurate lay observer and because some two to three weeks later a young man, twenty years old, living in a house some fifteen to twenty rods from the pasture where these calves ran, was taken sick with poliomyelitis with resulting paralysis of the lower extremities.

The other instance of the disease in lower animals, to which I referred, occurred at a farm in the town of Essex where there were two cases of the disease in the same family.

To quote the language of Dr. C. M. Ferrin, health officer:

“The father of these two boys bought some pigs from a party who had, as it transpired, pigs in his herd that had some paralytic disease of the hind legs. The pigs which this father bought developed a similar paralysis and were fed and cared for by the boys. Within two or three weeks from the time these pigs became affected the boys were taken sick.”

These boys, one after the other, had poliomyelitis, being taken sick two days apart.

It is of course regrettable that neither of these instances of disease in the lower animals was discovered in time to have adequate pathological or bacteriological examinations made. They emphasize, however, the facts: that the disease affects lower animals, that it occurs in connection with the disease in the human family, and that there are good *prima facie* reasons for thinking there may be a common cause for cases in man and the lower animals, and that it may be communicated from animals to man and vice versa.

The year 1910 was not a dry year.

The precipitation data for Vermont in 1910 compared with the means as observed at the Weather Bureau Station at Burlington are as follows:

	<i>April</i>	<i>May</i>	<i>June</i>	<i>July</i>	<i>August</i>	<i>Sept.</i>	<i>Annual</i>
Means,	1.98	3.01	3.34	4.14	3.60	3.56	32.68
1910	2.10	3.42	3.10	3.06	2.76	2.76	31.63

It will be observed from these figures that while the precipitation for the early summer months was somewhat below the means for this state, that the difference was not very marked. I may remind you, in this connection, that the precipitation for the year 1894, in which the epidemic previously referred to occurred, in Rutland County, was only 22.96, which is, with one or two exceptions, the least ever reported in the state. The annual precipitation for the year 1909 was 35.76; and for the year 1908, 23.49, the latter being also extraordinarily low. The year 1910, however, was not an exceptional year as far as the precipitation records show.

In conclusion, I wish to call attention to certain facts, bearing on the prevention of this disease.

First, "infantile paralysis" and "acute poliomyelitis" are perhaps more properly "epidemic poliomyelitis."

Second, the disease is infectious, and, to a certain extent, contagious. It can probably be transmitted by third persons and possibly inanimate objects, as well as by the sick. It seems to be equally a disease of man and animals.

Third, it apparently spreads from epidemic centers in the direction of the greatest travel.

Fourth, the discharges from the air passages, kidneys and bowels are all to be viewed with suspicion as possibly infectious.

Fifth, this disease may affect adults.

Sixth, it is a disease that is dangerous to life, as well as disastrous in its after effects on those who survive.

Inasmuch as there are various grades of severity of the disease, which we recognize by the paralysis, there are undoubtedly mild abortive cases. It is impossible except with laboratory facilities for animal experimentation at the present time to diagnose these abortive cases with any certainty. Febrile and grippy cases with vague nervous symptoms of any description occurring in a neighborhood where there are cases of poliomyelitis, should be treated by health officials as cases of that disease.

Health officials and physicians should be on the alert during the summer months to recognize this disease as early as possible. They should report any suspicious paralytic disease in any of the lower animals to the State Board of Health. Every scrap of information bearing on this disease in man, as well as the lower animals, should be carefully gathered and sifted in order that every community may contribute as much as possible to the solution of the cause of this increasingly prevalent infection.

Perhaps I cannot do better than to quote from the circular recently issued by the American Orthopedic Association and the American Pediatric Society and addressed to health authorities and boards of health:

"All cases of infantile paralysis should be strictly quarantined, sputum, urine and feces being disinfected, the same rigid precautions being adopted as in scarlet fever. This quarantine should, in the opinion of the committee, last for four weeks in the absence of definite knowledge as to when the infection ends. Children from infected families should

not be allowed to go to school until the quarantine is abandoned. The transportation or transfer of acute cases in public conveyances should be strictly forbidden. It would be very desirable to adopt provisional quarantine measures in suspicious cases in a community where an epidemic prevails. The report of all cases of infantile paralysis to the public health authorities should be enforced by law, and all deaths from this cause should be properly described and registered. A careful study of epidemics by public authorities is strongly advised."

To the health officers of Vermont let me say: Let every physician learn from you that this disease is reportable. Enforce the laws as to reporting diseases that are "infectious and dangerous to the public health" as regards poliomyelitis. Enforce the "full quarantine" in this disease. Disinfect and clean up after the acute stage is passed, as you would after diphtheria.

ANTERIOR POLIOMYELITIS IN VERMONT IN 1911*

BY CHARLES S. CAVERLY, M.D.

IT may be recalled that Vermont suffered a rather marked outbreak of epidemic poliomyelitis during the year 1910; it should be further noted that the 1910 outbreak affected chiefly the Connecticut and tributary valleys, and that the brunt of the epidemic was felt chiefly about Brattleboro, Windsor, Montpelier, Barton and Irasburg. In these regions occurred fifty-two of the seventy-two cases which were recorded in the state. This number includes three cases, records of which were received too late for tabulation in the Report for 1910.

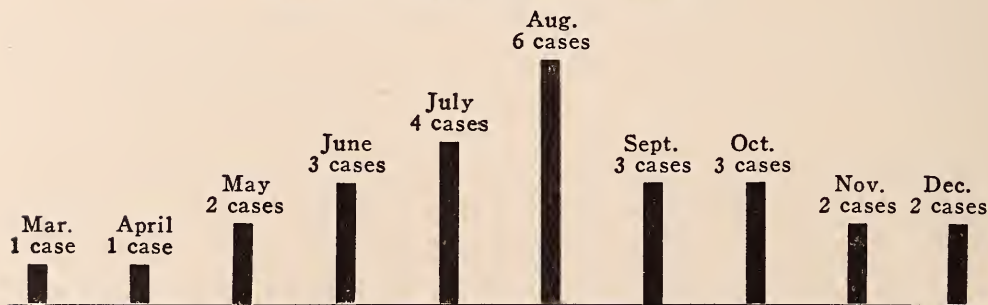
Of the twenty cases occurring on the western side of the state, the chief centers were Rutland and Burlington.

Records of twenty-seven cases have been obtained for the year 1911.

The disease in 1911 was distributed throughout the year, beginning in March. The hottest months show, as usual, the largest number of cases, but there is not the preponderance that is the rule.

POLIOMYELITIS. VERMONT, 1911. SEASONAL DISTRIBUTION.

VERMONT STATE BOARD OF HEALTH.



*Reprinted from *Bulletin of the Vermont State Board of Health*, Vol. XII, No. 3, Mar. 1, 1912.

Reference will be made presently to the relation which the disease bore to temperature and precipitation.

The geographical distribution of the disease in 1911 in Vermont is interesting. The following chart shows the distribution by counties:

DISTRIBUTION BY COUNTIES EAST AND WEST.

<i>East</i>		<i>West</i>	
Orleans	0	Rutland	16
Washington	1	Chittenden	0
Windham	0	Addison	3
Windsor	3	Lamoille	0
Caledonia	1	Bennington	2
Essex	0	Franklin	1
Orange	0	Grand Isle	0
Total	5	Total	22

A comparison of the geographical distribution of the disease in 1910 with that of 1911 shows that the eastern and western sides of the Green Mountains have changed places. In 1910, 72 per cent of the cases were on the eastern side of the mountains, while in 1911 over 81 per cent of the cases were on the western.

Orleans County, which had the largest share of the 1910 cases, had none in 1911, while Washington, Windham and Windsor Counties, which were conspicuous counties in 1910, had in 1911 only four cases.

On the western slope (Champlain Valley), the chief centers in 1910 were Rutland and Burlington. In 1911 there were no cases in the Burlington district, while the severity of the outbreak was confined quite closely to Rutland.

In this connection it must be remembered that the great outbreak of 1894 (the first large outbreak of this disease recorded) was in the valley of the Otter Creek. In 1910, fifteen of the twenty-two cases occurring in Western Vermont occurred in this valley.

An analysis of the population, physical characteristics, general sanitation and industries of the Otter Creek Valley is interesting in this connection. The portion of the valley affected is that just about the river in the towns of Rutland, Proctor and Pittsford.

The population is cosmopolitan. The industry is marble quarrying and manufacture. The river carries the sewage of the towns, is sluggish, except when broken by the falls at Center Rutland and Proctor. The rock formation is, of course, limestone. At Proctor, the focus of the 1911 outbreak, the population is probably more cosmopolitan than elsewhere in the state; this population is generally well housed, and the general sanitation is excellent. As compared with West Rutland, in which also marble is the chief and only industry, the general sanitary conditions in Proctor are superior and the quarrying population better housed. Yet there was only one case in West Rutland and that at a point remote from the quarries and mills. The chief obvious difference in the two towns is the fact that Proctor is in the Otter Creek Valley, while West Rutland is not.

In the absence of exact knowledge of the infecting organism of poliomyelitis and its methods of spreading, any deductions from the above facts are perhaps superfluous. We may theorize to the extent of guessing that the stream (Otter Creek) has something to do, directly or indirectly, with the spread of the disease.

The towns affected in 1911 were as follows:

TOWNS HAVING DISEASE

Cavendish	1 case.
Pawlet	1 case.
Proctor	9 cases.
Pittsford	2 cases.
Rutland	3 cases.
West Rutland	1 case.
Enosburg	1 case.
West Burke	1 case.
Middlebury	1 case.
Vergennes	1 case.

Weybridge	1 case.
Manchester	2 cases.
Cabot	1 case.
Norwich	1 case.
Hartford	1 case.
Total	27 cases.


The distribution of the disease in proportion to the population is shown by reference to the table following:

<i>East</i>	
Washington	1 to 41,702
Windsor	1 to 11,229
Caledonia	1 to 26,031
<i>West</i>	
Rutland	1 to 3,009
Addison	1 to 6,670
Bennington	1 to 10,689
Franklin	1 to 29,866

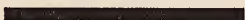




The map herewith attached will give a graphic picture of the geographical distribution of the disease in the state.

The two following charts show the age and sex of the cases.

DIVISION OF CASES BY SEX.

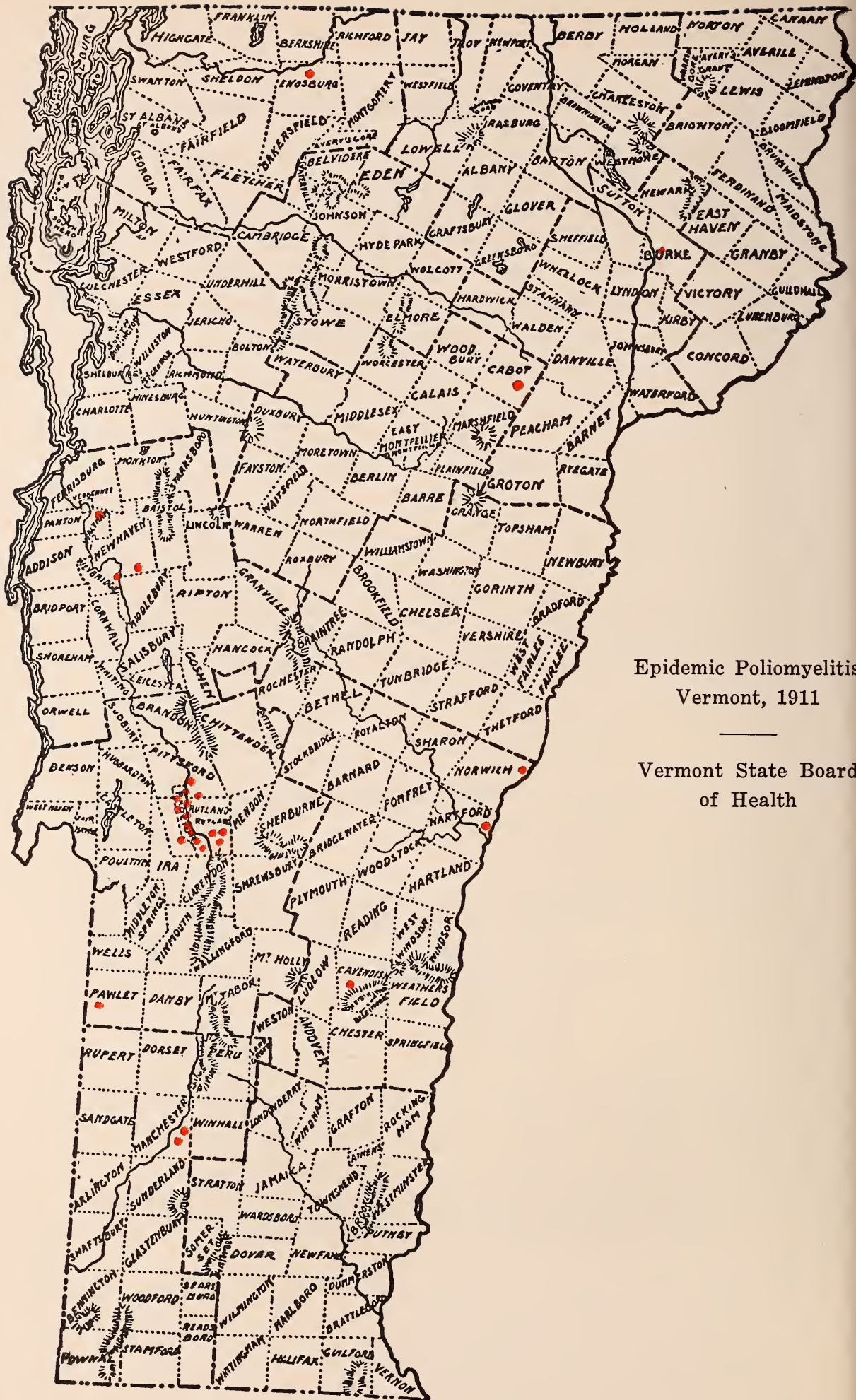
Male	18	
Female	9	

DIVISION OF CASES ACCORDING TO AGE.

Under 5		12
5 to 10		4
10 to 20		8
20 to 30		2
30 to 40		1
40 to 50	None	

The usual preponderance of the male sex is emphasized this year, 66 per cent of the cases being male.

AGE: Under 5 years, 12 cases or 44 per cent approximately.
Between 5 and 10 years, 4 cases or 14 per cent approximately.
Between 10 and 20 years, 8 cases or 29 per cent approximately.
Over 20 years, 3 cases or 11 per cent approximately.



Epidemic Poliomyelitis
Vermont, 1911

Vermont State Board
of Health

The figures for this outbreak as to age limits show a preponderance of cases under 10 years, but not as great as is usually the case.

The average age of all these cases was 10 years and 7 months; the average age of those who died was 11 years, and of those who survived, 9 years, 11 months.

The nationalities affected were:

American	16
Anglo-Saxon	1
Canadian	1
Hungarian	2
French	2
Swedish	2
Scotch	1
Not stated	2
Total	<hr/> 27

Other facts about the surroundings of the patients are as follows:

AGE OF HOUSE	
Under 10 years	3
Over ten years	22
Not stated	2

DETACHED HOUSE OR TENEMENT HOUSE	
Detached house	15
Tenement house	7
Not stated	5

SOIL ABOUT THE HOUSE	
Dry	17
Wet	6
Not stated	4

PROXIMITY OF POSSIBLE INFLUENCES	
Steam railway within 50 rods	7
Street railway on street	7
Stream or pond within 50 rods	15
Manufacturing establishment within 50 rods ..	8

Domestic animals, especially cats, horses and hens, were generally either kept by patients' families or were kept in the immediate neighborhood. There were no authenticated instances of coincident paralytic disease in any domestic

animals this year. It may be stated, however, that a hen paralyzed in its legs and one wing, furnished by Dr. G. G. Marshall of Rutland, July 26, was examined pathologically by Dr. B. H. Stone, director of the Laboratory of Hygiene, with negative results.

There were no recognized cases in the human family, however, within a half mile at least of the place where this hen sickened and the earliest Rutland case was August 24.

Some attempt was made to secure facts about the presence of vermin in the affected houses and the general sanitary conditions within and in their immediate vicinity. Nothing of an exact or reliable nature was secured along these lines. It is, however, a safe statement, based on general observation, that the sanitary conditions (cleanliness) of many of the infected premises were poor. There were, however, notable exceptions to this rule.

WATER SUPPLY

Well	2
Public aqueduct	14
Spring	9
Not stated	2
Total	<hr/> 27

In this connection it may be stated that the water supply of Proctor is a mountain stream, the intake being about seven miles from the village. The affected families in that village all received this supply.

SEWER FACILITIES

Public sewer	17
Dry closet	4
Private sewer	3
Not stated	3

CASES LOCATED IN RAILROAD TOWNS

Number of cases that occurred in a town located on a railroad ..	24
Number of cases that occurred in towns off the railroad	3

FAMILY HISTORY

Good (No case of chronic disease, like tuberculosis, cancer, diabetes, Bright's disease or rheumatism in immediate family.)	19
Fair (One case of such disease only in immediate family.)	5
Bad (Two or more such cases in immediate family.)	1
Not stated	2

ASSIGNED CAUSES

Among the assigned causes by attending physicians might be mentioned: a boy of 12 years, who was attacked immediately after attending a circus and drinking freely of so-called "birch beer and circus lemonade."

Another, too, a boy of 16, who worked in a hot dry-house and who was obliged to go into the cold November air frequently to carry lumber in connection with his work.

Several of the cases, as is usual under these circumstances, had been accustomed "to go in swimming" during the hot weather.

Eight of the cases are known to have had large tonsils or adenoids or both.

Information was sought as to the number of cases that had been away from home within a month and only seven such cases were found. None of these, however, had been, as far as could be ascertained, where there had been cases of poliomyelitis.

PREVAILING DISEASES IN TOWN DURING YEAR

In towns where diphtheria prevailed there were	7 cases.
In towns where measles prevailed there were	4 cases.
In towns where scarlet fever prevailed there were	3 cases.
In towns where chicken pox prevailed there were	3 cases.
In towns where whooping cough prevailed there was	1 case.
In towns where smallpox prevailed there was	1 case.
In towns where no disease prevailed there were	7 cases.

The early symptoms noted were as follows:

EARLY SYMPTOMS NOTED

Fever in	23 cases.
Headache in	12 cases.
Pain in	17 cases.

Tenderness in	11 cases.
Vomiting in	10 cases.
Constipation in	9 cases.
Twitching in	8 cases.
Delirium in	6 cases.
Chills in	5 cases.
Diarrhoea in	3 cases.
Head cold in	3 cases.
Convulsions in	2 cases.

The symptoms during the attack are shown in the following table:

SYMPTOMS DURING ATTACK

Disturbances of digestion in	22 cases.
Disturbances of bladder in	13 cases.
(7 of the 13 retention.)	
Pain and tenderness in	13 cases.
Pain general in	6 cases.
Pain in extremities	7 cases.
Pain in neck and back	4 cases.

The day in which the paralysis appeared was as follows:

DAY OF DISEASE PARALYSIS OCCURRED

1 to 3 days	16
3 to 7 days	5
After 7 days	1
Not stated	5

The distribution of the paralysis in the non-fatal cases was as follows:

Both legs	5
One leg	5
One arm	3
Both legs and one arm	2
Arm and leg (same side)	1
Both arms	1
One side of face	1
All extremities	1

The initial paralysis in the fatal cases is shown in the following chart:

Both legs	3
One leg	1
All extremities	1
Arm and leg (same side)	1
One arm	1
No data obtainable	1

Summary of conditions of cases 6 to 9 months after attack:

CONDITION OF CASES SIX TO NINE MONTHS AFTER ATTACK

Fully recovered	5
Some degree of paralysis	13
Died	8

A striking fact about this year's outbreak is its high mortality. The highest mortality which any recent figures show, as far as I have observed, is that in Pennsylvania in 1910.* In a series of 1076 cases the mortality was 22 per cent. The common figures for mortality in this disease are about 8 to 12 per cent.

The disease was of a very virulent type in 1911. Twenty-nine per cent of the cases recorded were fatal. The distribution of the fatal cases is interesting. They were distributed as follows:

Rutland	2
Manchester	1
Vergennes	1
Enosburg Falls	1
Cabot	1
Cavendish	1
Burke	1

It will be noted that there were no deaths in Proctor, where there were one-third of all the cases in the state. The so-called sporadic (or scattered) cases seem to have been generally fatal.

The mortality in the state last year was 14 per cent. This year (1911) it is twice as great.

A further analysis of the fatalities from poliomyelitis in Vermont in 1911 recalls an interesting fact regarding the possible seasonal influence on the virulence of the infection, viz. all the fatalities during that year occurred in the colder months.

*Drs. Dixon, Karsner, "Epidemiologic and Etiologic Studies of Acute Poliomyelitis in Pennsylvania."

In March there was 1 case with 1 death.
 In April there was 1 case with 0 death.
 In May there were 2 cases with 0 death.
 In June there were 3 cases with 0 death.
 In July there were 4 cases with 0 death.
 In August there were 6 cases with 0 death.
 In September there were 3 cases with 1 death.
 In October there were 3 cases with 2 deaths.
 In November there were 2 cases with 2 deaths.
 In December there were 2 cases with 2 deaths.

Total, 27 cases with 8 deaths.

No deaths were recorded during the hottest summer months, when most of the cases occurred.

The mortality during this year by age periods was as follows:

Of 12 cases under 5 years	2 died or	17 per cent.
Of 4 cases from 5 to 10 years	2 died or	50 per cent.
Of 8 cases from 10 to 20 years	2 died or	25 per cent.
Of 2 cases from 20 to 30 years	1 died or	50 per cent.
Of 1 case from 30 to 40 years	1 died or	100 per cent.

29.6 per cent of reported cases died.

Referring to these reports, as regards the records of possible connection of one case of the disease with another, one interesting case of this kind should be mentioned.

A young man employed in a postoffice (cancelling stamps) was attacked in September. His place, after an interval of about one week, was taken by another young man. This latter worked only a week at the same work as the former. In less than a week after he had given up this work he was taken sick with this disease and died in about three days.

These boys had no special association before or after their employment in the postoffice and there were no other cases in the town.

The postoffice where they successively worked is a new building. The room was not kept clean, and there was considerable dust in the air. A water closet opening into this room was quite filthy, due to careless use.

MONTHLY TEMPERATURE, 1911.
BURLINGTON WEATHER STATION.

	<i>Mean</i>	<i>Normal</i>
March	24.0	27.3
April	40.2	40.7
May	62.5	53.9
June	62.8	63.8
July	72.5	68.2
August	68.4	66.1
September	56.4	58.9
October	46.7	46.9
November	34.0	33.7
December	31.0	22.5

These temperature records show that May and December were much above the normal mean. July was also a hot month. The mean for the year (44.4) was 1.4 degrees higher than normal.

PRECIPITATION

The precipitation records of the Weather Bureau for Vermont show a general deficiency for the year 1911.

By months they are:

	<i>Amount</i>	<i>Normal</i>
March	2.44	1.83
April	0.83	1.87
May	1.13	2.83
June	2.54	3.26
July	2.47	3.78
August	3.83	4.01
September	3.46	3.35
October	2.84	3.16
November	1.56	2.58
December	2.51	1.69
Year	26.52	31.56

These figures show a dry summer. Corresponding figures for five years are:

	<i>Amount</i>
1907	29.67
1908	23.49
1909	35.76
1910	31.63
1911	26.32

No decided advance has been made, during the past year, in our knowledge of the exact cause of poliomyelitis or of methods of treating or preventing it.

Flexner and Clark have confirmed, by recent animal experiments, the fact that the virus is present in the tonsils and pharyngeal mucosa of human beings who succumb to the disease.

Hence the care that should be constantly exercised in cleansing the naso-pharynx in cases of the disease and in destroying all discharges from nose and throat.

Health officers and physicians should always remember that the disease is contagious and quarantinable.

In the summer, and especially during the presence of known cases in a community, physicians should be alert to the possibility of "abortive" cases. Children and young adults who present "grippy" or "rheumatic" symptoms, or who have any vague febrile attack should be regarded with suspicion.

To the physicians of the state who have aided in securing the data herewith presented, our thanks are due.

The same investigations will be pursued during 1912.

EPIDEMIC POLIOMYELITIS (INFANTILE PARALYSIS) IN VERMONT*

By CHARLES S. CAVERLY, M.D.

PURSUING the plan undertaken, when poliomyelitis again appeared in epidemic form in Vermont in 1910, the State Board of Health has collected during the last biennial period such data as were available in regard to this growingly important disease. Dr. H. A. Ladd, the Inspector of this Board, has collected most of the data on which the reports for these years are based. These reports deal with the years 1912 and 1913, and continue the reports of the years 1910 and 1911, published in the last biennial report of the State Board of Health.

The blanks used for collecting the data here presented are those recommended by the U. S. P. H. S. and certain data for comparison have been taken from "Epidemiological Studies of Acute Anterior Poliomyelitis" by Surgeon Wade H. Frost of this Service.

It is not supposed that the following statistics include all cases of poliomyelitis that have occurred in this state during the biennial period. There have, without doubt, been cases of the "abortive" type of this disease, in connection with each local outbreak.

During 1912, there was no epidemic of this disease. Most of the 13 cases, however, occurred along old epidemic trails in the valleys of the Connecticut and Otter.

Following somewhat the form of report made in the last two years, the incidence of the disease by months was as follows:—

*Reprinted from Bulletin of the Vermont State Board of Health, Vol. XIV, No. 4, June 1, 1914.

POLIOMYELITIS—VERMONT—1912

Seasonal Distribution*Geographical Distribution*

In 1910, the majority of the cases in this state occurred on the East side of the Green Mountains, in the Connecticut Valley; in 1911, on the West side, on the Otter-Champlain watershed.

In 1912, the cases were about evenly divided between these valleys.

POLIOMYELITIS—VERMONT—1912

*Geographical Distribution**EAST (Connecticut Valley) Counties*

Total, 8 cases

WEST (Champlain Valley) Counties



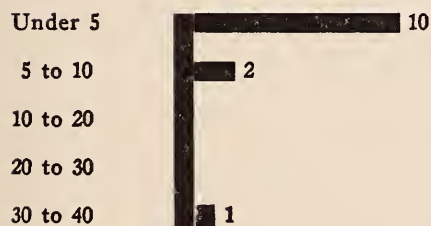
The accompanying map shows the distribution graphically.

There were 8 males and 5 females in this series.

Age

The age distribution of these cases was as follows:—

POLIOMYELITIS—VERMONT—1912
Ages



The preponderance of cases under 5 years is, of course, not exceptional.

Nationality

American	8
Canadian	3
Polish	1
Italian	1

The occupations of the family wage-earner were:

Laborer	5
Farmer	4
Granite cutter	1
Electrician	1
Glazier	1
Butter maker	1



Poliomyelitis
Vermont, 1912
Vermont State
Board of Health

The general sanitary surroundings of patients' houses were:

Good	4
Fair	7
Bad	2

Toilet facilities were:

Water closets	7
Earth closets	6

The other data regularly sought with reference to this disease, such as character of the house (age, tenement or detached), soil conditions, proximity of factories, railroad, streams, etc., domestic animals kept, water supply, family history, personal history, presence of infectious diseases in the towns affected, first symptoms noted during the attack, revealed nothing extraordinary in this year. The cases occurred, like the old sporadic cases, lacking epidemic proportions, though mostly at old epidemic centers.

Day on which paralysis occurred:

1st	2
2nd	3
3rd	3
4th	1

Distribution of paralysis:

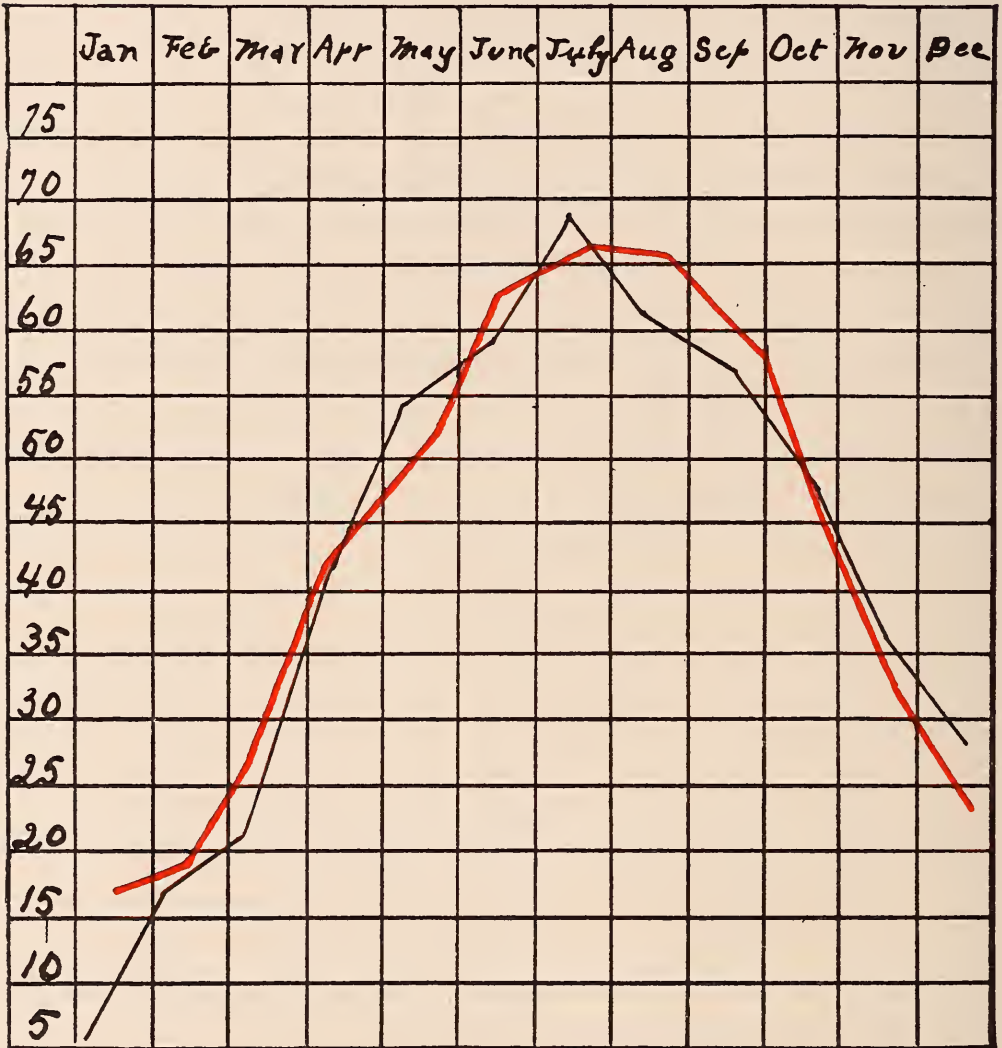
All extremities	5
Both legs	3
One arm and one leg (same side)	2
One arm and face	1
One leg	1
Both arms	1

The severity of these cases is indicated by the extent of the paralysis; also by the fatalities:

Permanently paralyzed	4
Complete recovery	4
Died	5

This, of course, is an exceptional mortality. The mortality in scattered cases has usually been high in this state. Perhaps this is somewhat due to the overlooking of mild or abortive cases.

MEAN MONTHLY TEMPERATURES, BURLINGTON WEATHER BUREAU, 1912, COMPARED WITH NORMAL.



Normal (33 years), red line —————

Monthly means (1912) black line —————

The mortality under 5 years was	30%
The mortality between 5 and 10 years was	50%
The mortality over 30 years was	100%

The average age of those who died was 8 years. Three of the 5 deaths occurred in cold months, viz.: December, February and March.

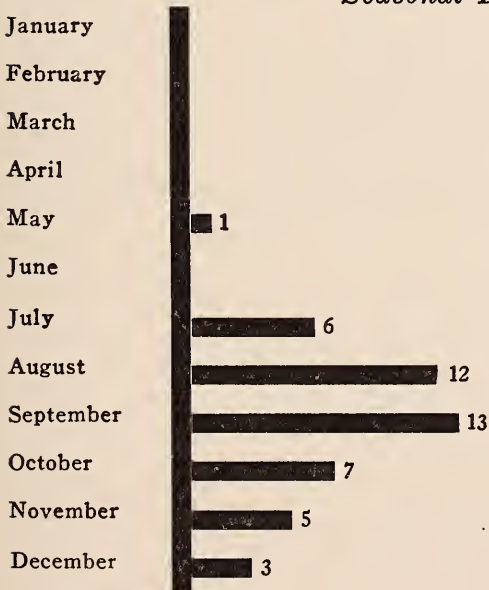
No direct or indirect contact with the infection was traceable in 10 cases. In the other 3, contact was probable or certain. In one fatal case, a subsequent history was obtained of a probable abortive case in the family two or three weeks before. One other instance of two cases in the same family occurred.

- 2 cases occurred in families with 1 other child.
- 5 cases occurred in families with 2 other children.
- 4 cases occurred in families with 3 other children.

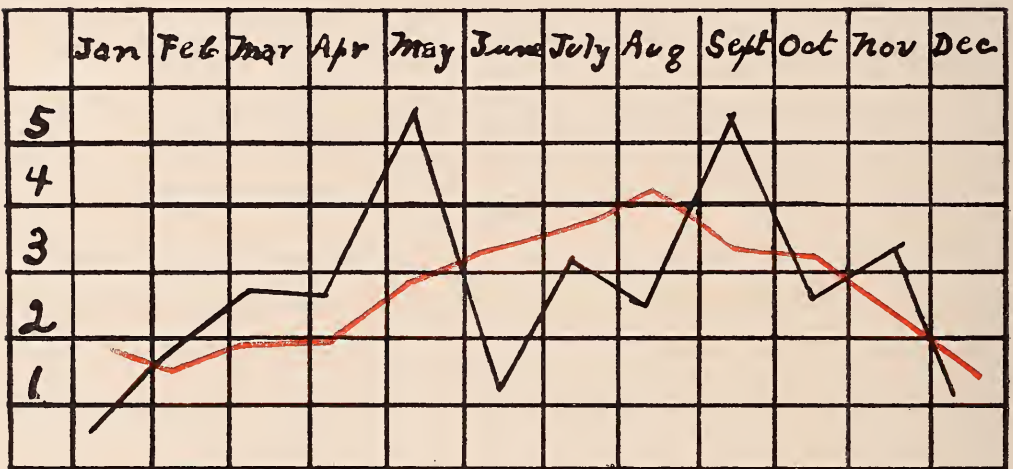
1913

The number of cases of poliomyelitis in the state took a sharp rise again in 1913. The disease assumed epidemic proportions in a section of Caledonia and Orleans Counties, centering about Hardwick. The features of the disease as it prevailed in the state are briefly as follows:

POLIOMYELITIS—VERMONT—1913
Seasonal Distribution



MONTHLY PRECIPITATION IN INCHES COMPARED WITE
NORMAL, 1912, BURLINGTON WEATHER
BUREAU FIGURES.



Normal (35 years) red line —————

Monthly totals (1912) black line —————

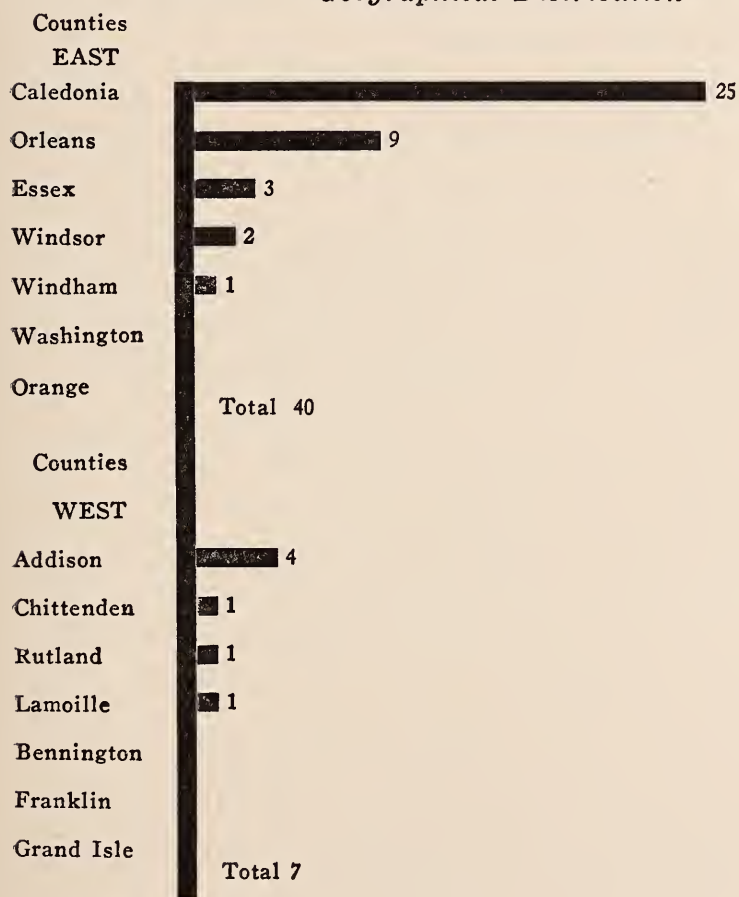
Total precipitation for the year 34.14 inches.

Mean precipitation for this locality, 31.56 inches.

While the *summer* months of 1912 were dry, the precipitation *for the year* was rather above the normal.

The epidemic in the northeastern section of the state started in July. The first case was in Hardwick, occurring on July 12th. The patient, who worked in a stoneshed more or less, was the son of a section hand on the railroad. (This patient had had tonsils and adenoids removed seven years before.) The epidemic in the Hardwick section culminated in September. Five of the July cases in the State occurred at Hardwick. The sixth occurred in Burlington—an isolated case. The scattered cases in Walden, Wheelock, Lyndon, Barton and other towns occurred during the months of August, September and October. Glover cases occurred in October and November.

POLIOMYELITIS—VERMONT—1913

Geographical Distribution

The geographical distribution of poliomyelitis in Vermont from year to year is very interesting. The last marked outbreak of the disease in the state in 1910 showed 51 cases on the east side of the Green Mountains, in the Connecticut Valley, and 18 on the west, in the Champlain Valley. In 1911, the west side of the state had 22 cases to 5 on the east, while in 1912, with only 13 cases reported in the state, there was no epidemic center and the cases were divided more evenly, 8 and 5.

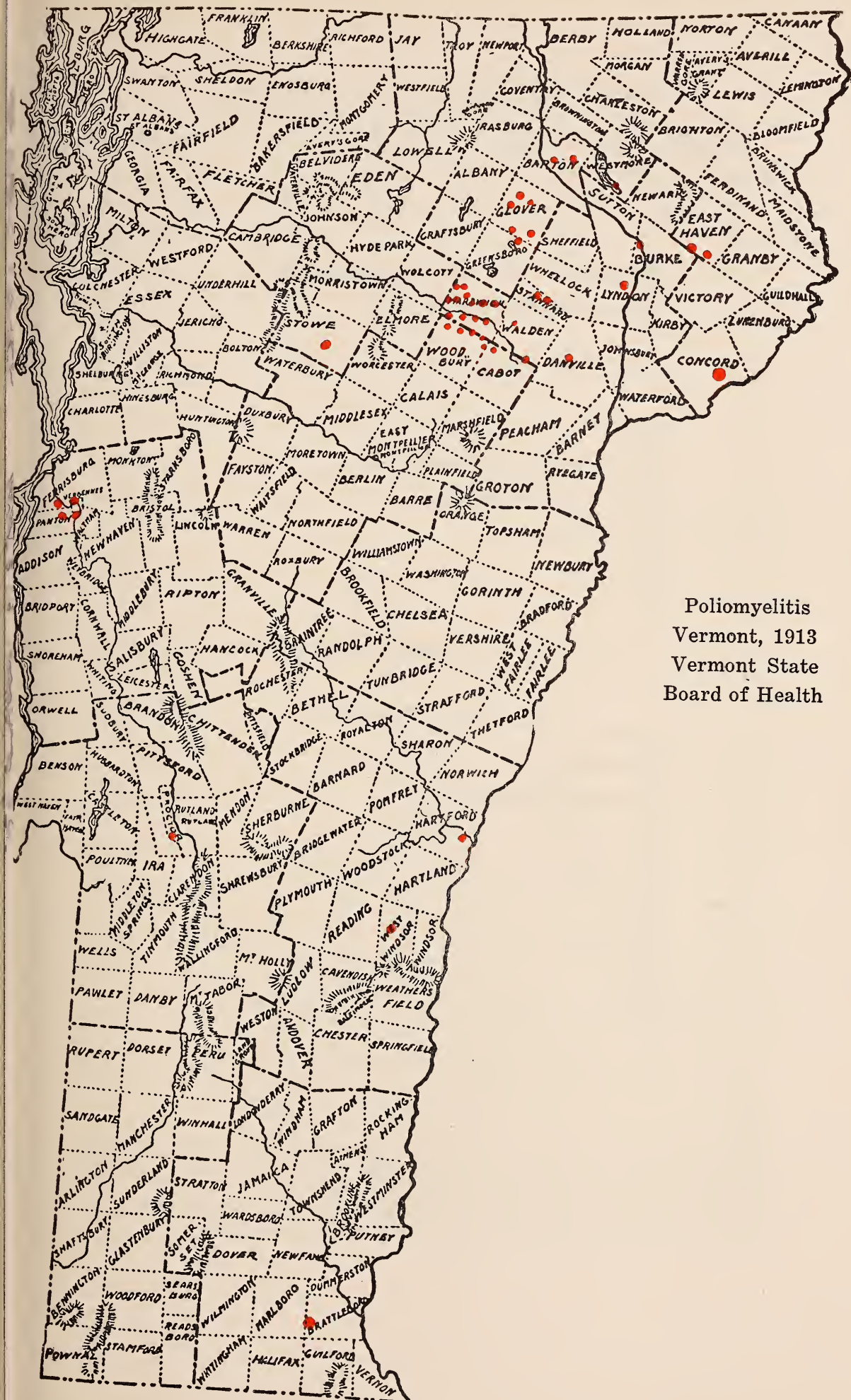
Of the 47 cases reported, 37 can fairly be assigned to the Hardwick outbreak. There was one other interesting and rather striking outbreak (4 cases) at Vergennes. Occurring late in the season, the first case in the series occurred on November 30th in the child of a liveryman. The disease was not recognized for some time and the possibility of stomoxys carriage was overlooked.

The per capita occurrence of poliomyelitis in 1913 by counties and towns, where the epidemic cases occurred, was as follows:—

Caledonia County	1 case to 1041 population
Essex County	1 case to 2461 population
Orleans County	1 case to 2593 population
Addison County	1 case to 5002 population
Hardwick town	1 case to 188 population
Glover town	1 case to 155 population
Vergennes City	1 case to 371 population

These figures throw light on the seriousness of the outbreak in the towns and counties chiefly affected. Orleans County was the epidemic center of the 1910 outbreak; the adjoining county of Caledonia had the greatest number of cases this year. The starting of the epidemic, as has been mentioned, was in Caledonia County, and the cases in the adjoining counties of Essex and Orleans, very likely, in some way owed their origin to those in Caledonia.

Reference to the map, showing the geographical distribution of the disease in the state in 1913, will show at a glance the clustering of the cases in this region.



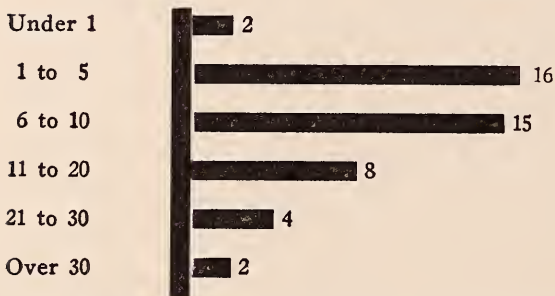
Poliomyelitis
Vermont, 1913
Vermont State
Board of Health

The following charts show the division of our cases this year by sex and age:—

POLIOMYELITIS—VERMONT—1913

Sex

POLIOMYELITIS—VERMONT—1913

Age

The day on which the paralysis appeared in these cases was as follows:—

Day Paralysis Appeared

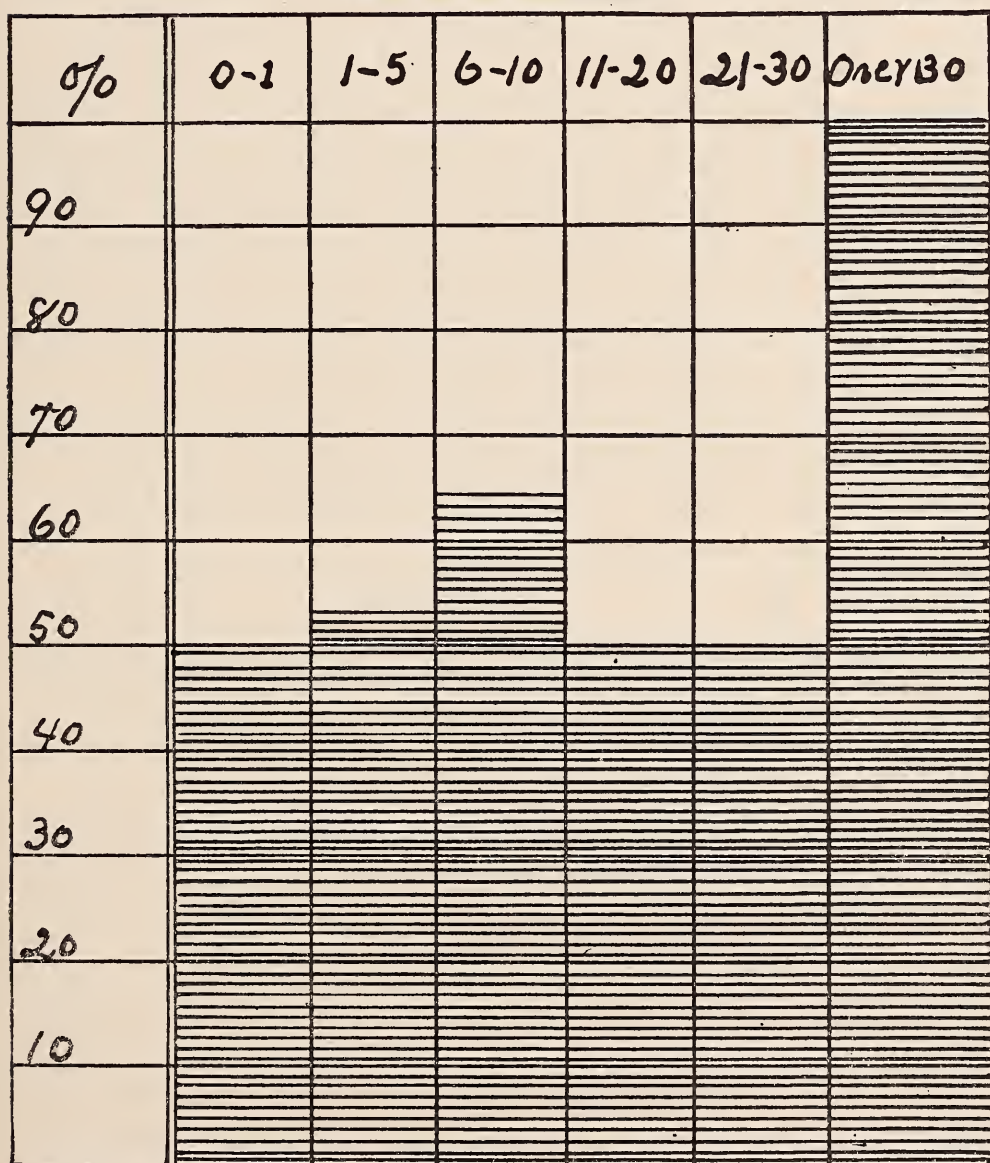
This chart shows nothing unusual, the preponderance of cases in this disease showing paralysis on the 2nd to 4th day.

It has been noted by several observers that the relative proportion of males increases after adult life. The relative number of cases in males and females, as a whole, in Ver-

*Ratio of Males to Females Reported in Certain States
in Recent Epidemics of Poliomyelitis*

Epidemic		Total Cases	Males		Females	
			Number	per cent	Number	Per cent
Massachusetts	1909	620	363	58	263	42
Minnesota	1909	332	193	58	139	42
Iowa	1910	345	200	58	146	42
New York	1910	227	118	52	109	48
Dist. Columbia	1910	246	151	61	95	39
Vermont	1910	69	40	57	29	43
Vermont	1913	47	28	59	19	40

Chart showing the relative number of males and females, at different
ages, in Vermont, 1913.



Shaded spaces, Males.

Blank spaces, Females.

mont, in 1913, showed about the average preponderance of males. By age groups, however, the equal division in the second decade was somewhat unusual.

Distribution of Paralysis

One leg	12 cases
Both legs	14 cases
One arm	3 cases
Both arms	2 cases
Leg and arm (same side)	5 cases
Both legs and arm	3 cases
Both legs and both arms	3 cases
Facial only	3 cases
Deglutition	1 case
Died before noted	1 case

The involvement of the muscles supplied by cranial nerves in this disease is not unusual. Three cases in which the only noticeable paralysis was one side of face and one of the muscles of deglutition is perhaps a rather large proportion of 47 cases. Two of these cases occurred in the apparently isolated outbreak of four cases at Vergennes—one-half the whole number in that outbreak.

The results in these cases, as far as can be determined March 1, 1914, are indicated in the following chart:—

Results to March 1, 1914



There was no attempt made to identify or classify abortive cases, in the essential outbreak of which this is chiefly a report. That there were such cases in connection with the main epidemic in Hardwick cannot be doubted. Several frank cases of paralysis in this outbreak occurred in families and neighborhoods where other children had presented vague febrile or “grippy” symptoms. The lateness, however, with which these suspicious cases were seen and their

present normal condition threw so much uncertainty on the nature of their symptoms that they are not considered in these statistics. Several paralyzed cases were certainly known to have been in contact with such doubtful cases.

The sister of one adult case had symptoms, which pointed to an abortive attack of poliomyelitis, two weeks after the paralyzed case was taken sick. The symptoms she suffered were pain and weakness of legs and temporary loss of reflexes. This apparently abortive case is not included in these statistics.

The elimination from consideration of the abortive cases throws most of our cases into the "still paralyzed" column. Only one of the whole group is known now to have fully recovered, and this was a cranial nerve case with facial paralysis.

Forty-two of these forty-seven cases, then, still show some degree of paralysis. This, in a few cases, could perhaps be better described at this time as a weakness of a leg or group of muscles.

Recovery of function, however, in this large proportion of cases has not yet been complete.

The next notable fact about this year's outbreak is the low mortality, viz., 8.5 per cent.

- Of 2 cases under 1 year, 50 per cent were fatal.
- Of 16 cases between 1 and 5 years, 12.5 per cent were fatal.
- Of 15 cases between 6 and 10 years, 0 per cent were fatal.
- Of 8 cases between 11 and 20 years, 0 per cent were fatal.
- Of 4 cases between 21 and 30 years, 0 per cent were fatal.
- Of 2 cases over 30 years, 50 per cent were fatal.

Two of the fatal cases occurred in connection with the main epidemic in and about Hardwick. The other two were apparently isolated or sporadic cases. It was observed (see Report of State Board of Health of Vermont, 1910 and 1911) in the outbreak of poliomyelitis in this state in 1911, that all the deaths occurred in the cooler months (March,

September, October, November and December) and that none occurred in connection with the chief epidemic.

In 1913, of the four deaths, one occurred in May, not connected apparently with any epidemic center, one in August, an isolated case, and the other two in July and August, in connection with the Hardwick-Glover outbreak.

An interesting coincidence (probably only this) was connected with the sickness and death of one adult—an isolated case, apparently. This man's brother had died of the disease two years before in a hospital in New York State. There was no connection probably between the cases, although some of the clothing worn by the Vermont patient, prior to his illness, had formerly belonged to the brother in New York. The latter, however, had not been in contact with it after his illness began. This same patient, ten days prior to his illness, had spent an hour or two at a relative's house, in another Vermont town, where a young man had died of this disease twenty-two months before.

One of the Hardwick cases was taken sick on the same day that a sister at their home, in a town twenty miles distant, was taken with the same disease. The Hardwick cases had been in Hardwick only two days. This suggests that the origin of the disease in these children was the same. No contact with a prior case was discoverable, either direct or indirect.

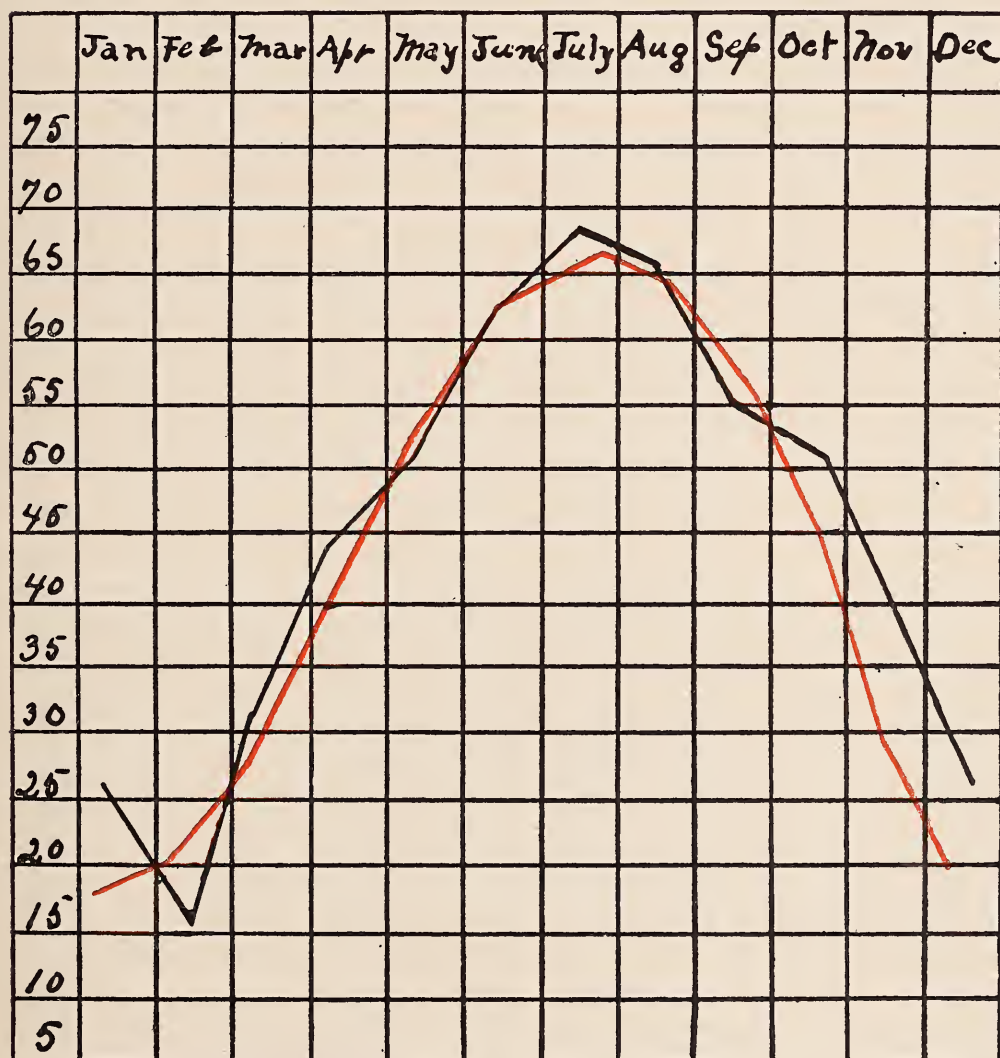
The symptoms shown in these cases during the acute stage were uniformly those common to the disease: fever, vomiting, pains in joints and extremities and constipation were quite regularly present.

Respiratory failure was usually the cause of death, in fatal cases.

The tabulated statement of the possible contact cases in this outbreak follows:—

History of probable contact within 2 weeks	8
History of certain contact within 2 weeks	6
No history of contact within 2 weeks	33

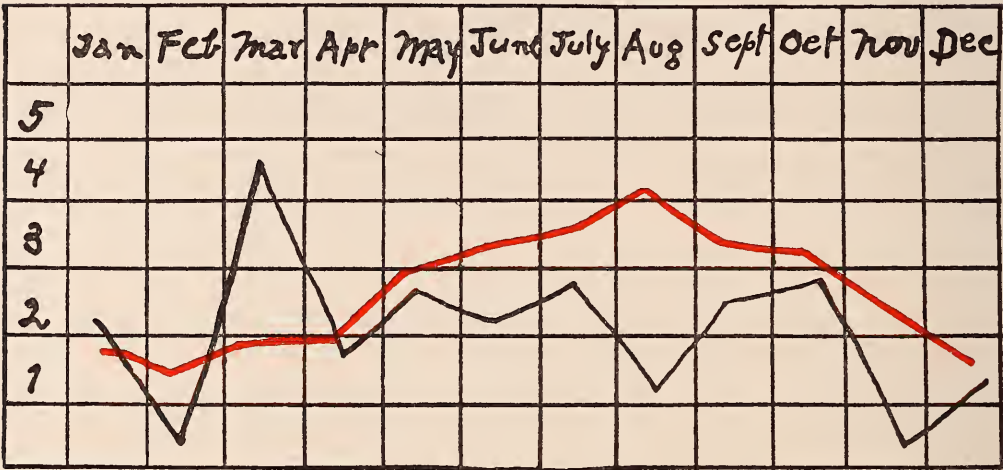
BURLINGTON WEATHER BUREAU CHART,
SHOWING MEAN MONTHLY TEMPERATURES IN VERMONT, 1913,
COMPARED WITH NORMALS.



Normal (33 years) red line. —

Monthly means (1913) black line. —

MONTHLY PRECIPITATION IN INCHES AT BURLINGTON
WEATHER BUREAU, VERMONT, 1913,
COMPARED WITH NORMAL.



Normal (35 yrs.) red line. —————
Monthly totals (1913) black line. —————

From these charts it will be seen that the summer of 1913 was about normal as to temperature, but dry. The total precipitation for the year, 25.75 inches, was over 8 inches below the normal for this region. The dry season was very apparent. It was especially noticeable in Hardwick and vicinity. Streams were dry, also springs, and the village water supply in that town failed. The Lamoille River, which flows through the village, was at its lowest, with scarcely a perceptible stream. Drains and sewers emptied on its bank and into its nearly dry bed, and the village streets were unsprinkled. Flies were correspondingly numerous.

The same natural conditions, of course, obtained elsewhere.

GENERAL FACTS ABOUT THE CASES

Nationality		
American	33
Canadian	5

Italian	3
Spanish	3
English	1
Irish	1
Scotch	1

Chief Occupation of Parent or Wage Earner

Farmer	10
Laborer	7
Stone cutter	8

The remaining 22 cases were scattered among 20 occupations.

The occurrence of epidemic poliomyelitis in our state in the neighborhood of stone industries has been noted before. Hardwick is a granite-cutting town. Of the 17 cases occurring in that town, 8 were in the families of stone cutters. While Barre, the home of large granite industries, has heretofore largely escaped visitations of this disease, Montpelier, Rutland, West Rutland and Proctor, centers of large stone industries, have suffered. Indeed, the first great outbreak of this disease in this country, in 1894, occurred in Rutland County and was centered about its marble works. No special significance, has, so far as known, hitherto been attached to this coincidence of stone working and poliomyelitis. The Hardwick outbreak, however, after the experiences of these other stone-working towns, is noteworthy. The connection, of course, may be sought in local sanitary defects, or habits of the men who are engaged in stone working, rather than in anything inherent to the work itself.

General Sanitary Conditions

These conditions are classified thus:—

Good	18
Fair	20
Bad	9

The toilet facilities in houses occupied by cases were:—

Flush closet	23
Privy	23
Cess pool	1

Previous health of patients for a month prior to the attack:—

Good	41
Poor	6

It may be noted that none of these cases were ascribed to injuries, to bathing in streams or other bodies of water in hot weather, or to other infectious diseases.

Large tonsils or adenoids in these cases were common. Those whose antecedent histories were described as “poor” had usually vague gastro-intestinal disturbances, or chronic weaknesses, or were described as “delicate children.”

Occurrence of Multiple Cases in Same House or Family

Of the 47 cases, here recorded, 37 were single cases in a family or house. Six of these single cases occurred in families where there was one other child.

- 14 occurred in families with 2 other children.
- 3 occurred in families with 3 other children.
- 5 occurred in families with 4 other children.
- 1 occurred in family with 5 other children.
- 2 occurred in families with 6 other children.
- 1 occurred in family with 7 other children.

This once more emphasizes the rather slight contagiousness of the disease, as there is little likelihood that any of these other children escaped contact with the sick, after the disease developed.

There were five instances of two cases in the same family.

Occurrence of Cases on Traffic Routes

Thirty-six of the cases occurred in railroad towns, 11 in towns away from the railroad.

Hardwick is on a cross-state railroad. It is not a trunk line road or one with much traffic from neighboring states. In this respect it differs from the epidemic centers of former years, like Brattleboro, Windsor, Barton, Rutland and

Burlington. The first case at Hardwick was in the family of a railroad section hand. This case occurred July 12. The next two cases occurred six and seven days later respectively. One of these two cases was on an isolated farm some two miles from Hardwick village, the other in the village. No known contact was traceable between these latter and the first case.

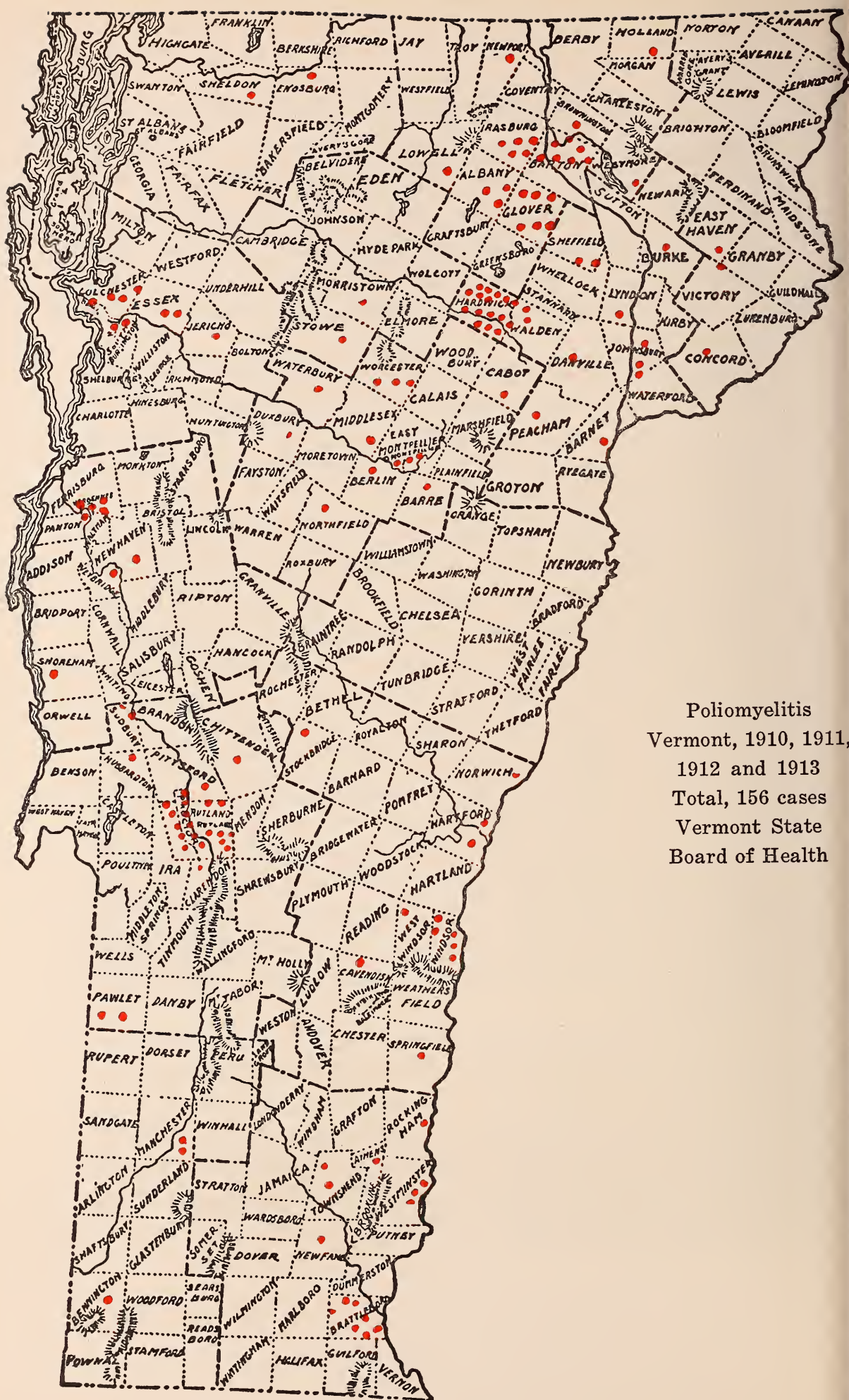
Glover and Wheelock, not railroad towns, are on or near a thoroughfare much travelled by teams and automobiles, and it is noteworthy that the cases in these towns and Barton occurred either late in the Hardwick outbreak, or afterwards, in October or November.

That this disease follows lines of human traffic, rather than the valleys, through which sewage-polluted rivers flow, is quite obvious by a glance at the map. None of the Lamoille Valley towns, below Hardwick, had a case of the disease. This, in spite of the fact that the railroad follows this valley nearly to its end.

While this year's outbreak in this state has not been as extensive, or the cases as numerous as has happened in some former years, the intensity of the outbreak in Hardwick has been unprecedented in recent years in Vermont. This record includes only such cases as were paralyzed. Vague and questionable cases, sometimes probably real "abortive" cases, have been carefully excluded. This for the obvious reason that with our present facilities for diagnosis, there must be some question always about the genuineness of these cases.

It should be stated that several experiments were made on monkeys in connection with the foregoing cases by Dr. B. H. Stone, Director of the Laboratory.

In one adult case in 1912, 5 c. c. spinal fluid was taken by lumbar puncture and injected into frontal lobes of cerebrum of Rhesus monkey. The fluid was transparent and the result was negative.



Poliomyelitis
 Vermont, 1910, 1911,
 1912 and 1913
 Total, 156 cases
 Vermont State
 Board of Health

Bedbugs taken from the bed in which the above case died were emulsified and introduced into the frontal lobe of a Rhesus monkey— results negative.

Swabs from the throats and noses of eight cases and contacts in Hardwick were rubbed over the throats and noses of Rhesus monkeys without results.

Watery emulsions of flies, taken from three infected premises at Hardwick, were injected into the cerebrums of Rhesus monkeys—all negative.

POLIOMYELITIS IN VERMONT DURING THE FOUR YEARS 1910-1913

A review in brief of our experiences with this disease in Vermont during the past four years is interesting. A cursory glance at the accompanying map will show that the bulk of the cases in this state have occurred in a few localities. Of the 156 cases reported in this state during this quadrennium:

7 cases have occurred in Brattleboro, five of which were in one year (1910).

24 cases have occurred in or near Rutland, 15 of which occurred in one year (1911).

6 cases have occurred in or near Windsor, 5 of these in 1910.

8 cases have occurred in or near Burlington, 7 in 1910.

8 cases have occurred in or near Vergennes, 4 in 1913.

13 cases have occurred with Montpelier as the center, 8 in 1910.

65 cases have occurred in the northeastern counties of Orleans, Caledonia and Essex, centering about Hardwick and Barton.

37 of these occurred during the outbreak of 1913, and 24 occurred in 1910. These epidemic centers are all in larger river valleys with the exception of Barton; are all on main

traffic lines except possibly Hardwick, located on a cross-state railroad.

A casual survey of the map for the four-year period shows the clumping of red dots about the centers mentioned, all on main thoroughfares through the state or on the larger streams. It should perhaps be repeated that the occurrence in the main valleys (on large streams) is very evidently due to the stream of human intercourse that takes these paths.

This latter conclusion is emphasized by the almost complete absence of red dots on the map along the more mountainous portion of the state, north and south. That there are other influences at work, determining the epidemic incidence of this disease, than lines of human intercourse, is certain. Hardwick, somewhat off the main beaten path of railroad traffic, has had perhaps the severest visitation from this disease of any town in the state in recent years. Why this town, rather than St. Johnsbury, or Newport, or St. Albans, all on main railway lines, should suffer, is probably to be answered by a search of local conditions. Exactly what these were, we may be able to answer sometime when the etiological factors in the spread of this disease are better known. Low rain fall, dust, filth of all kinds, perhaps the consequent multiplication of insects, must all be considered. The infection was imported into Hardwick in some way, found a fertile soil in some local condition, and multiplied accordingly.

SUMMARY

The foregoing completes such epidemiological studies as we could make of poliomyelitis in Vermont for the past four-year period. Only cases frankly paralytic have been included in our figures.

The number of cases has fluctuated in such a way as to point to a three-year period for the recurring epidemic move.

1910	69 cases
1911	27 cases
1912	13 cases
1913	47 cases

It is hoped that continued epidemiological observations of this disease in our state may indicate any possible periodicity about its recurrence in the state as a whole, on the two sides of the Green Mountains (in the Connecticut and Otter-Champlain Valleys) as well as at the evident epidemic centers of the disease in the state.

Doctors Flexner, Clark, and Amoss, in one of the most recent publications from the Rockefeller Institute, record some experiments in monkeys with the virus of the disease, which show that a strain of poliomyelitic virus was propagated four years; during this time it showed three distinct phases: first of low virulence, then of maximum and finally a return of the low virulence. They say "The cycle of changes in virulence is correlated with wave-like fluctuations in epidemics of disease, which also consist of a rise, temporary maximum and fall in the number of cases prevailing." It will be interesting to know if field observations of the periodicity of poliomyelitis outbreaks correspond with the results obtained experimentally. The same report of these animal experiments contains this, relating to the possible explanation of obscure outbreaks in remote localities:—

"In the light of this presentation the part played by sporadic and abortive cases and of the microbe carriers of potentially epidemic diseases becomes more comprehensible. We may consider this class of infected persons or animals as carrying specific micro-organisms lacking high virulence for their respective kind. We may begin to see how the conversion, through favoring causes, of micro-organisms of low into others of high virulence, may be the signal for the appearance of epidemics, not necessarily confined to one

place, but possibly arising almost simultaneously in separated and even remote places when the conditions are similar; just as, on the other hand, the immediate transportation of already elevated micro-organisms from a place in which an epidemic is already prevailing to new places may start similar severe outbreaks there."

No rational or scientific explanation has yet been proposed to explain the sudden appearance of this disease in isolated country districts. Such cases, as is well known, occur in epidemic seasons and at other times. The disease is as apt to attack small children, who may never or seldom have been away from home or apparently had any communication whatever, for weeks before the attack, with the outside world.

We may hope that the experiments mentioned above may help to solve the perplexing problem of the transmission of this disease, the origin of these obscure (sporadic) cases and the difference in virulence of individual outbreaks. While experimental proof has been adduced for the stable fly (*Stomoxys*) and bed bug theories of the carriage of this disease, these experiments have not been uniformly positive.

The experiments, above referred to, of Dr. Flexner and his assistants, point to human agencies (the sick and "carriers") as the chief factors in the spread of infantile paralysis. From these experiments we may infer an explanation of "abortive" or mild cases.

Observed facts about the epidemiology of this disease have never fitted laboratory theories. If these recently reported experiments, showing the transition of organisms of low to high virulence, and then to low again, in animals, holds good in the human species, these experiments may prove highly important to a better understanding of the mysterious occurrence of cases of this disease.

The whole trend of medical thought of late in regard to methods of dissemination of infectious disease has been

towards human rather than fomites, or animal, agencies. Contact with the frankly sick, convalescents, "missed" cases and "carriers" explains most of our cases and outbreaks of infectious diseases. Poliomyelitis may prove no exception to this rather general rule.

The worst outbreaks here, as elsewhere, have not been in industries, rather significantly. As previously pointed out, this may be only a coincidence, the true significance lying in local conditions, or the character and habits of the workers themselves.

Vermont is interested in the discovery of the cause of this disease, inasmuch as the state has suffered more than most from its ravages. For this, as well as other reasons, the State Board of Health will continue its epidemiological investigations of the disease and earnestly solicits the continued help of the *medical profession, as well as local health officials*.

SUGGESTIONS TO HEALTH OFFICIALS, PHYSICIANS AND OTHERS IN REGARD TO EPIDEMIC POLIOMYELITIS

1. Poliomyelitis is a *reportable* disease. In the presence of an outbreak cases of sickness in children with vague nervous symptoms should be viewed with suspicion. Such symptoms are headache, convulsions, unusual general weakness, or weakness of a limb or group of muscles. In some outbreaks, grippy symptoms, sore throats, headache and general pains are quite common premonitory symptoms. The so-called "abortive cases" may end with these, and yet be as dangerous to others as frankly paralyzed cases.

Practitioners are cautioned to be watchful for such cases, during the season of poliomyelitis (June to September). They should be reported and investigated. The statute requires reports of *known* cases and of *suspicious* cases.

2. This disease is *quarantinable*. The *full* quarantine should be enforced four weeks. It is known that the disease

is *infectious*, the infection is known, and it is communicable—although probably communicable to a limited extent. Every precaution must be adopted against its spread, in view of the disastrous results of every outbreak.

3. Special attention should be given to the *noses* and *throats* in *cases* and in those in *contact with cases*.

It is known that the nose and the throat may harbor the germ of the disease and that the germ may reach the brain and cord via these passages.

Therefore the use of a 1 per cent solution of peroxide of hydrogen as *gargle* or *nasal wash* is best for cases and contacts. All excreta, especially all *nasal* and *mouth discharges* from cases should be disinfected. *Heat* is the safest disinfectant.

4. In the presence of the disease, *insects* (especially flies) should be carefully excluded from the sick room and house. Filth of all kinds, especially stable manure, should be removed and disinfected, the breeding places of flies destroyed. *Street* and *house dust* should be laid with water or oil.

5. *Terminal disinfection* should be as thorough as in all diseases subject to the full quarantine.

6. Do not forget that the disease may attack *adults*; and that it may occur in *cold weather*.

EPIDEMIC POLIOMYELITIS

A REVIEW OF THE EPIDEMICS OF 1914 AND 1915*

By CHARLES S. CAVERLY, M.D.

VERMONT has had an unusual experience with this disease. In the summer of 1894, the first considerable outbreak that had been reported occurred in this state. This outbreak, comprising 132 cases, was confined almost exclusively to the Valley of the Otter on the west side of the Green Mountains.

From 1894 to 1910 nothing approaching an epidemic of this disease appeared in the state. In the latter year, following a rather severe outbreak in the Connecticut Valley in Massachusetts, the same valley in Vermont was invaded. Of 69 cases occurring that year, 51 occurred on the east side of the Green Mountains.

This disease was made a reportable one at that time under the regulations of the State Board of Health. The "Full Quarantine" was enforced in these cases. This quarantine is the same as is practiced in the state with other major infections, like scarlet fever and diphtheria.

The disease, without doubt, has been as fully reported since 1910 as was possible with the imperfect knowledge we have of its diagnostic points. The profession of the state has been generally aware of the possibility of local outbreaks, especially in rural communities, and has cooperated very generally with the State Board of Health in identifying these cases and applying preventive measures.

*Reprinted from "Bulletin of the Vermont State Board of Health," Vol. XVI, No. 4, June 1, 1916.

In 1911 there were 27 cases reported in the state, 22 of these were on the west side of the Green Mountains and 19 of them in the same valley in which the original epidemic of 1894 occurred.

In 1912, there were only 13 cases reported in the state and these were quite widely scattered with no epidemic focus. Eight of these were in the Connecticut Valley and 5 on the west side of the Green Mountains.

In 1913, 47 cases were reported in the state. Forty of these occurred in the northeastern section of the state, and 37 in Hardwick and its immediate vicinity. It should be noted that the last cases in this 1913 outbreak occurred in the town of Barton, only about 25 miles from Hardwick. Since 1910, the Passumpsic-Barton Valley in Caledonia and Orleans Counties has furnished the chief foci of this disease in Vermont.

1914

The epidemic of 1914, in the number of cases, as well as in mortality, was the severest that has ever occurred in the state. The epidemic began in the Village of Barton, the first cases occurring respectively on July 9, 18, and 22. The first cases occurred in the immediate neighborhood in Barton Village, where the last cases in the outbreak of 1913 occurred in the month of November. No other cases occurred in the state, as far as our records go, until the 30th of July, when the first case occurred in Burlington. The earliest cases in Addison and Franklin Counties were respectively August 16 and August 14.

The outbreak of 1914 was chiefly felt in the northern half of the state above the forty-fourth parallel of latitude. Nearly 90 per cent of the 306 cases of which we have reports, occurred north of this line, which passes through Middlebury and Bradford. Scattering cases only occurred in Rutland, Bennington, Windsor and Windham Counties.

The medical practitioners of Vermont, generally alert to

the seriousness of this disease, had had most unusual opportunities for studying its various phases. The cases of the disease are undoubtedly diagnosed and reported by the profession with increasing thoroughness.

The following blank, which is a modification of the form recommended by the United States Public Health Service, was used in collecting most of the data this year, although the regular U. S. P. H. blank was used at first in Barton:

VERMONT STATE BOARD OF HEALTH

Case Report of Acute Anterior Poliomyelitis

Town
 Date of onset
 Date of paralysis (if any)
 Patient's name, age, Sex
 Nationality of father, of mother
 Occupation of father, of mother
 Residence (post office), county
 Did patient live in city?, village?, country?
 If in country, state distance from center of nearest town or village
, name of village
 If this is the first case in your town, state distance to R. R. station
, to livery stable, to a factory or stone or
 wood-working shop
 How near a stream, a pond, a lake
 Status of family: Well-to-do, moderate, poor
 General sanitary conditions: Excellent, good,
 fair, bad
 Previous general health of patient: Excellent,
 good, poor
 Tonsils: Large, unhealthy, normal
 Has patient adenoids?
 Had patient suffered from any illness, indisposition, or accident with-
 in a month prior to this attack?
 Nature of illness or accident

Other Members of Family, Including Guests, Boarders and Servants

CHILDREN:

Males (age of each)
 Females (age of each)

ADULTS: Males, number, females, number

Were there any other cases of sickness in the family within one
 month before or after this attack?

Give name, age, sex, date, and nature of each case

Symptoms of Acute Stage

Fever: High, moderate, slight, none
 Headache: severe, moderate, slight, none
 Constipation, diarrhoea, vomiting,
 sore throat
 Pain, distribution
 Tenderness, distribution
 Retraction of head, restlessness, drowsiness
 Any bladder symptoms
 State whether paralyzed or abortive case
 Distribution of paralysis at its worst

 What treatment was employed, and with what apparent results?
 (a) In acute stage

 (b) Subsequent to acute stage

Outcome of Case to Date

Recovery (complete disappearance of paralysis)
 Improvement, extent of paralysis remaining

 Death, date
 If an *abortive* case, does the patient present any symptoms after the
 acute initial form subside?

Contact with Previous Cases

Had patient been associated with any previous case? with a sus-
 pected (abortive) case?
 give name, address, and date

 Had any member of the patient's family been associated with any
 previous case?; if so, state whether paralyzed or ab-
 ortive; give name, address, and date

 Did patient attend school?, where, grade
 Has patient been to any large public gathering?; if so, place
 and date
 What were the weather conditions immediately preceding this attack?
 Hot, mild, cold, wet
 dry, dusty, unusual in any respect

 Have any infective diseases, respiratory or digestive troubles been
 unusually prevalent in the community?
 What animals or fowls are kept on the premises?
 What insects are noted on the premises?
 Has there been any paralysis of animals in the vicinity?

What preventive measures were carried out?
 REMARKS.— Please state any other facts of interest concerning the
 case

Date of filling out report

Signed, M.D.
 Address

Most of the cases were reported on these blanks by attending physicians or the inspector of the State Board.

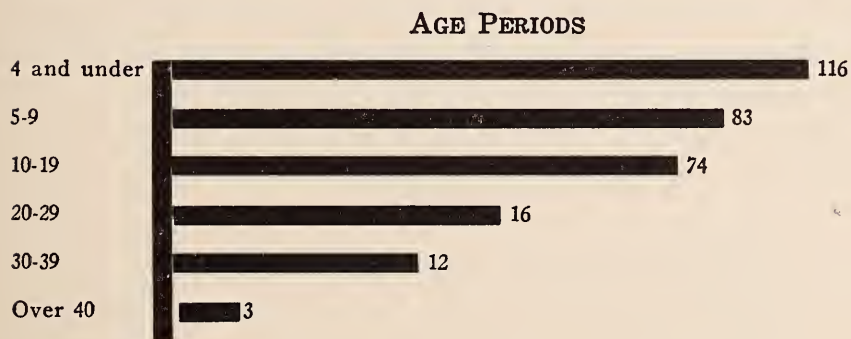
INCIDENCE OF THE DISEASE BY AGE AND SEX. 1914

Of the 304 cases* recorded in this outbreak, in which age and sex are given, 167 were males and 137 females.



The division of the cases by sex this year does not differ materially from the usual experience in these outbreaks. During the outbreak of 1910, the relative percentages were 57 for males and 43 for females; in 1913, 59 per cent males and females 41 per cent; this year the percentage is divided, males 55 per cent and females 45 per cent.

The occurrence of the disease by age periods was as follows:



The combined distribution of the cases in the various counties, by age and sex, was as follows:—

	4 & under		5-9		10-19		20-29		30-39		40 & over		Total
	†M	F	M	F	M	F	M	F	M	F	M	F	
Addison	4	7	3	6	5	3	1	0	2	0			31
Bennington	1	1											2
Caledonia		1			1				1				3
Chittenden	15	14	9	9	13	6	2	4	0	4			76
Franklin	8	6	4	6	3	4	1	1	2	0			35

*Two cases were reported later making the total number of cases 306.

†M Male; F Female

	4 & under		5-9		10-19		20-29		30-39		40 & over		Total
	M	F	M	F	M	F	M	F	M	F	M	F	
Grand Isle	2	3	1	0			1	0			1		8
Lamoille	6	2	3	2	3	1	1	0					18
Orange	0	3	4	5	2	4			1	0			19
Orleans	14	8	9	3	8	7	3	1	1	1			55
Rutland	5	3	4	1	3	1							17
Washington	4	3	4	6	3	4	1						25
Windham						1							1
Windsor	3	3	3	1	2	0					1	1	14
Total	62	54	44	39	43	31	10	6	7	5	1	2	304

DAY ON WHICH THE PARALYSIS APPEARED

The following table indicates the date of onset of the paralysis in those in which this detail is given. It will be noted that the paralysis appeared in this series of cases apparently a little earlier than has been observed in previous epidemics. While it is possible that errors have crept into the records in this respect, by reason of the failure of parents to note early symptoms in small children, it is a fact that in a considerable number of cases the appearance of the paralysis was the first intimation the family had of the illness of the child.

ONSET OF PARALYSIS

1st day	53 cases
2nd day	64 cases
3rd day	53 cases
4th day	43 cases
5th day	15 cases
6th day	10 cases
7th day	10 cases
After 7th day	9 cases

DISTRIBUTION OF THE PARALYSIS

The combinations observed in this series of cases of paralyzed muscles and groups of muscles were quite complex. In only a minority of the cases was the paralysis confined to one extremity, or a single group of muscles.

In the *fatal cases*, death was due to respiratory failure. Most of these cases were of the ascending type of paralysis,

all four extremities, or one or more of these being paralyzed frequently a day or two in advance of the respiratory symptoms. In 18 of these cases, all of the extremities were paralyzed prior to the respiratory paralysis; in 5, both arms; in 7, one arm and one or both legs; and in 9, the arm and leg on the same side of the body. Several of these cases showed paralysis of the muscles of deglutition and the neck. The distribution of paralyzed muscles in the cases that *remained paralyzed*, or that finally recovered without permanent paralysis, was extremely varied. It is impossible to classify in tables the parts involved except in a very general way:

All the extremities were wholly or partially paralyzed in	19 cases
Both arms in	9 cases
Left arm alone in	8 cases
Left arm and both legs in	6 cases
Left arm and left leg in	3 cases
Left arm and right leg in	5 cases
Right arm alone in	10 cases
Right arm and both legs in	6 cases
Right arm and left leg in	5 cases
Right arm and right leg in	9 cases
Facial paralysis alone in	12 cases
Both legs alone in	65 cases
Left leg alone in	30 cases
Right leg alone in	28 cases

The remaining cases gave a great variety of paralyzed muscles and groups. One case had paralysis of the left arm and muscles of the neck; another, "one arm" and the facial muscles; still another had paralysis of the right arm and right leg and the muscles of the right eye. The abdominal and spinal muscles were occasionally involved also with the extremities.

The sixteen cases described as not paralyzed were cases usually in the families of frankly paralyzed cases, which exhibited symptoms very suggestive of the initial symptoms of poliomyelitis, like fever, vomiting, pains of back and extremities, etc.

A fact observed not infrequently, especially at Barton where such cases were of frequent occurrence in conjunction with paralyzed cases, was this—in visiting and examining a paralyzed case, one or more children in the family would be noted as not acting quite well. In questioning the parents of such children, it was learned that they had had acute febrile attacks, usually within ten days or two weeks, and had not fully recovered their strength. The appearance of these children was very striking, as compared with others in the same family who had not had such symptoms. They were appreciably pale, weak and listless.

That, in all the communities where this disease appeared, there was a large number of such cases of varying degrees of severity, who recovered without any noticeable paralysis, there can be no doubt. These so-called abortive cases are surely important features of all outbreaks of infantile paralysis. The early diagnosis and control of these must have undoubtedly an important bearing upon the prophylaxis of epidemic poliomyelitis.

The records of this epidemic show that there were ten cases of crossed paralysis, *i.e.*, one arm and the opposite leg, and 12 cases of the hemiplegic type. One case was observed in which the eye muscles of one eye alone were involved.

The results of this outbreak of 1914, as determined by our reports dated from one to six months after the initial symptoms, were as follows:

Paralyzed cases	226
Died	53
Fully recovered	27
(Including those "not paralyzed")	

The mortality, therefore, in this outbreak was 17.3 per cent. The percentage of deaths by age periods was as follows:

PERCENTAGE OF DEATHS

By Age Periods

Of 116 cases, 4 years or under	11.2% died
Of 83 cases, 5 to 9 years	10.8% died
Of 74 cases, 10 to 19 years	21.6% died
Of 16 cases, 20 to 29 years	43.7% died
Of 12 cases, 30 to 39 years	50.0% died
Of 3 cases over 40 years	66.6% died
Of 306 cases (total number)	17.3% died
In 2 cases age not stated.	

The figures confirm previous observations as to the relation of age to mortality in epidemic poliomyelitis. The older the subject, the higher the mortality.

FEATURES OF THE EPIDEMIC

The symptoms presented by these cases during the initial stage of illness were quite regularly fever, vomiting, more or less pains in joints and extremities, not unusually constipation and retention of urine. Pain and tenderness along the spine were very frequently noted. In a few cases, as has been before stated, the first symptom that attracted attention was the paralysis.

The following table gives the number of instances in which there were one or more children under 20 in the same family with a frankly paralyzed case:

OTHER CHILDREN UNDER 20 IN FAMILY

1 other child under 20 in family	55 instances
2 other children under 20 in family ...	51 instances
3 other children under 20 in family ...	36 instances
4 other children under 20 in family ...	27 instances
5 other children under 20 in family ...	19 instances
6 other children under 20 in family ...	13 instances
7 other children under 20 in family ...	4 instances
8 other children under 20 in family ...	1 instance
9 other children under 20 in family ...	3 instances
10 other children under 20 in family ...	1 instance

As bearing on the communicability of this disease, a careful tabulation of the known exposures to both abortive and frank cases gives this result in our 306 cases:

Instances in which no known contact could be traced with paralyzed, abortive or carrier case	238
Instances in which contact with a supposed abortive or carrier case was shown	31
Instances in which contact with a frank case was shown	37

The following notes bearing on house infection are also interesting:

In one family of nine children, three had "indigestion" with febrile symptoms seven to nine days before a frank case occurred.

In one family of six children, there were two paralyzed cases eleven days apart.

In another family of four children, there were two cases of frank paralysis seven days apart.

In another family of six children, there were two paralyzed cases, also seven days apart.

In another family of five children and two adults, two children were taken sick August 22 and 26, respectively, one case proving fatal, and the father, thirty-eight years old, was taken sick August 26, and was one of the severest cases in the whole outbreak that recovered, being paralyzed in all extremities.

INSTANCES OF ONE OR MORE CASES IN THE SAME FAMILY OCCURRED AS FOLLOWS

In family with 2 children under 20	5 instances of two or more cases in either parents or children or both
In family with 3 children under 20	5 instances of more than 1 case
In family with 4 children under 20	2 instances of more than 1 case
In family with 5 children under 20	2 instances of more than 1 case
In family with 6 children under 20	3 instances of more than 1 case
In family with 7 children under 20	2 instances of more than 1 case
In family with 9 children under 20	1 instance of more than 1 case
In family with 10 children under 20	2 instances of more than 1 case (1 paralyzed and 1 abortive)

One fatal case occurred in a family in which there was a case three years before.

Many instances occurred in histories that were elicited from these families of symptoms very suspicious of abortive cases in other children. Such cases were corroborated in some instances, as has been before noted, by the appearance of such children.

The above facts may or may not indicate a *communicable* disease. If, as seems likely, many "potential agents of dissemination"* occur in family as well as community-life under epidemic conditions, the disease may be classed as really communicable. The bare fact, however, of only one or two frank cases of the disease often occurring in large families, indicates on the surface slight contagiousness.

Prior Diseases

Of diseases preceding the acute stage of the disease, a great variety were given. There was, however, not one disease that could be positively given as a contributing cause. Grippy colds, gastro-intestinal diseases, attended with diarrhoea and a generally debilitated condition, were oftenest mentioned, but even these figure in only an insignificant number of cases.

There was one case undoubtedly mistaken for appendicitis and the appendix removed on September 9, followed by paralysis the following day.

Prior Injuries

There were, too, a considerable number of histories of more or less definite injuries antedating the paralysis.

One child with a paralysis of the left deltoid, arm and forearm had a fractured humerus ten days before on the same side. Another had a "broken arm" from seven to ten days before the attack, which proved fatal within forty-eight hours. In still another case, a fall and rather vague

*Simon Flexner, M.D. "Modes of Infection and Etiology of Poliomyelitis."

injury or strain of the right arm was followed in ten days by the disease and paralysis of that arm alone.

There were not the usual number of histories of "going in swimming," although a few were so reported. Six or seven cases of more or less vague injury to the head by falls and blows thereon were also reported.

Tonsils and Adenoids

Forty-four cases gave a definite history of enlarged or diseased tonsils.

Three cases had been operated on for tonsils and adenoids within three years.

OCCUPATIONS OF WAGE EARNERS, 1914

The 286 cases, in which the occupation of the breadwinner of the family is stated, were reported as follows:

Farmer	112
Laborer	50
Mechanic	16
Teamster	11
Stone-cutter	8
Stone-worker	8
R. R. employee	7
Army officer	6
Merchant	5
Painter	5
Plumber	5
Blacksmith	4
Mail carrier	4
Lumberman	3
Physician	3
Milk handler	3
Chauffeur	2
Manufacturer	2
Barber	2
Carpenter	2
Saloon worker	2
Town clerk	2
Telephone lineman	2
Miscellaneous (one each)	22

The condition of the premises and status of the family were reported as follows:

CONDITION OF PREMISES		
Excellent	64	
Good	104	
Fair	99	
Bad	37	
Not stated	2	
Total	306	

STATUS OF THE FAMILY		
Well-to-do	52	
Moderate	165	
Poor	82	
Not stated	7	
Total	306	

In connection with the foregoing tables, a statement of the nationality of the parents in this series of cases is instructive.

NATIONALITY			
<i>Father</i>		<i>Mother</i>	
American	193	American	202
Canadian	74	Canadian	68
Irish	10	Irish	7
English	8	English	5
Russian	3	Scotch	4
Not stated	8	Swedish	3
Scotch	2	Russian	3
Swedish	2	German	1
German	2	Italian	1
Italian	2	Bohemian	1
Bohemian	1	Not stated	11
Negro	1		

No effort has been made to summarize the reports in regard to domestic animals, insects or vermin on premises. This epidemic occurred under distinctly rural surroundings, with the possible exception of the cases which occurred in Burlington. Cats, dogs, cows, horses and hens are the regular accompaniments of all such premises. The unreliability of figures we have heretofore been able to collect in regard to insects and vermin have made any deductions from such statistics worthless. The epidemic occurred during "fly season." The presence of flies of several species was almost universal.

Paralytic Diseases in Animals

Instances of paralysis among domestic animals have always been noted as accompanying our outbreaks of human infantile paralysis. There were a few such instances in connection with this outbreak of 1914.

Instances of hens paralyzed on same farm	5
Instances of paralysis in cows or calves	3
Instances of paralysis in pigs	2
Instances of paralysis in dogs	2

DISTRIBUTION

Seasonal

The distribution of the cases during this year is shown in the following chart. The epidemic, as has been suggested, began in July, increased in August, reaching its climax in September, subsiding thereafter through the year.



It is only necessary to add that the seasonal curve of our cases in 1914 closely follows those of former years. August, September and October are the epidemic months in Vermont.

Geographical

While the southern half of the state is divided north and south by the Green Mountains, the same barrier does not

prevent intercourse between the east and west sides of the state in the northern half. As has been pointed out, the outbreaks in previous years affecting the southern part of the state have been quite largely confined to either the west or the east side of this mountain barrier and have shown some tendency to alternate between.

The 1913 outbreak focused about Hardwick. The outbreak of 1914 is traceable directly to that of the previous year, in that it started where the 1913 outbreak ended, viz., Barton. A reference to the map will show the widespread prevalence of the disease in 1914 over the northern half of the state. It will be further noticed that there seemed to be two main foci, Barton and Burlington. Noticeable clusters also occur in and about Bristol, Barre, Waterville and St. Albans. (See Chart I, page 116.)

The following table shows both the geographical and seasonal distribution of the disease. The division between the east and the west sides of the state is also given to conform to previous reports.

SEASONAL AND GEOGRAPHICAL DISTRIBUTION

County	<i>East</i>												Total
	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	
Caledonia								1	1	1			3
Orange								1	13	3	1	1	19
Orleans							7	32	11	4	2		56
Washington								5	12	7	1		25
Windham											1		1
Windsor								4	3	7			14
													118
County	<i>West</i>												Total
	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	
Addison								9	15	7			31
Bennington					1			1					2
Chittenden							1	25	44	4	2		76
Franklin								4	19	12	1		36
Grand Isle								1	4	2		1	8
Lamoille								4	14				18
Rutland		1						1	6	9			17
													188
Total	1				1		8	88	142	56	8	2	306

The river valleys, as well as the railroad and highways, in the northern half of the state run east and west as well as north and south, so that human intercourse and travel is far freer between the two sides of the state north of Burlington than south. This may explain, and without much doubt, does, the wide and comparatively even distribution of the cases over the northern half of Vermont.

Many questions might be raised with regard to the connection between the Barton and Burlington foci. A reference to the map (page 116) emphasizes the importance of these two centers of distribution of the diseases in 1914.

The outline map of the state (page 118), with some of the chief centers of the disease in 1914 marked in dotted lines, will show the sequence of the first cases in these centers. The first four cases of the outbreak, it will be noticed, occurred in Barton; the next two in Burlington; the seventh, in Middlesex; the eighth at Cambridge; and the ninth at St. Albans. The question of the connection between the early Barton cases and the first case at Burlington is an exceedingly important one, and one that, unfortunately, cannot be satisfactorily answered. Railroad and highway communication between Barton and Burlington is, of course, easily possible, but only via certain other towns and villages, which seem to have escaped. A railroad journey from Barton to Burlington necessarily would have to be broken at St. Johnsbury, or some other intervening point, and yet there was only a single case in St. Johnsbury, and that in the most rural part of the town. No other cases occurred at any other junction point between these places until some time later than the early Burlington cases. The tide of travel during the summer months through Barton is rather north and south than east and west. It would naturally be expected that carriers of this disease from Barton would distribute the infection into Canada, via Newport, or to the towns in the Connecticut Valley, via St. Johnsbury and

Wells River—this entirely on the supposition that railroad travel is a chief factor in the distribution of this infection. The ubiquity of the automobile and the facilities it offers for communication in all directions over highways must not be overlooked in this connection.

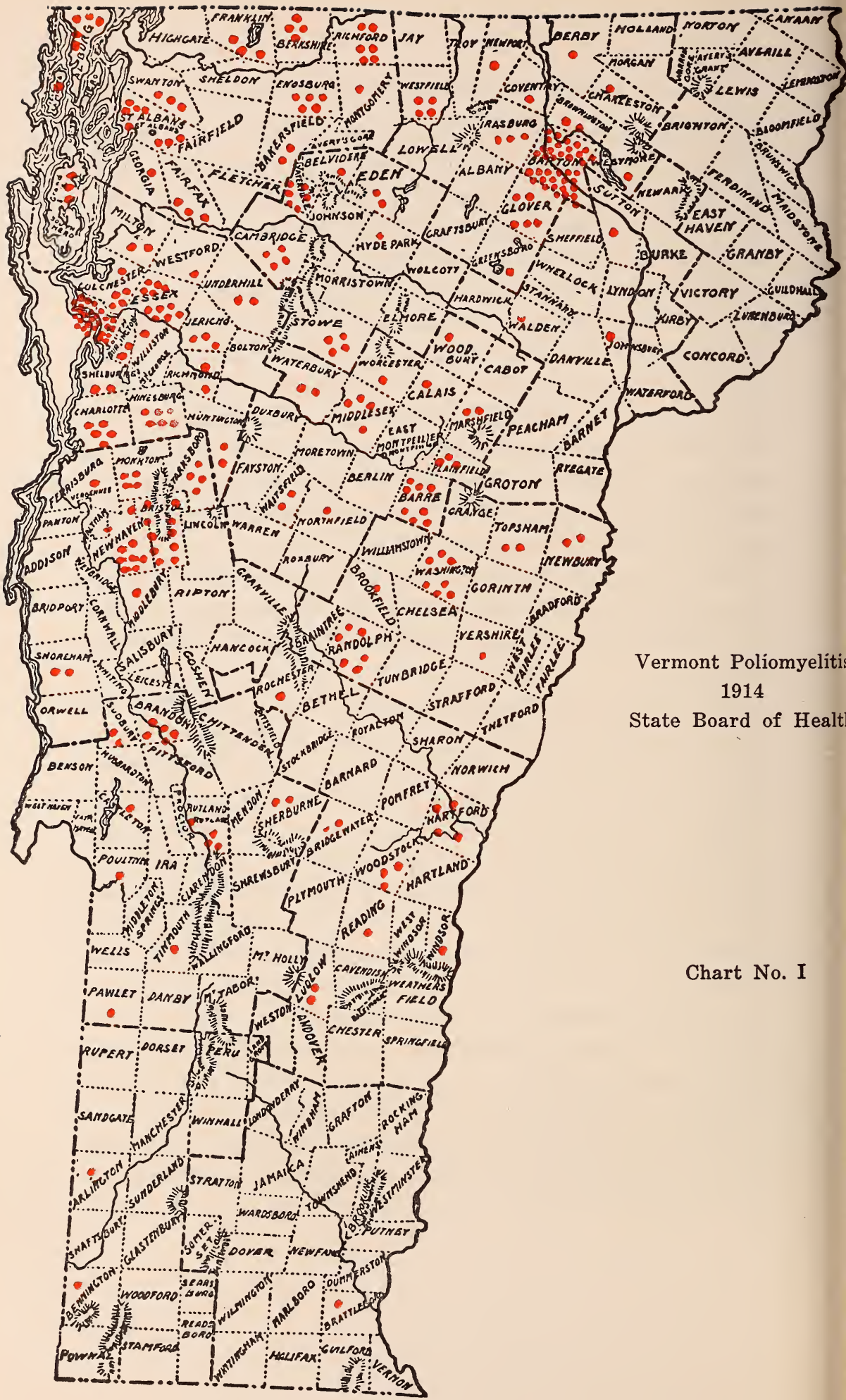
The part played by those “potential agents of dissemination,” abortive cases and human virus carriers in the distribution of this infection need not be enlarged upon; nor need we dwell on the fact, now generally recognized, that man is probably the chief distributor of all forms of disease organisms, and this includes the organism of infantile paralysis. The presumption, therefore, is that the Burlington focus, following that at Barton by about two weeks, owes its origin to the Barton center.

The Chart, No. VII, which shows the cases reported chronologically, by weeks, will demonstrate rather vividly the chronological relation of these two centers in this outbreak.

Given the connecting link between the Burlington and Barton centers, the local foci shown on Map No. II at St. Albans, Waterville, Middlesex, and Bristol (Charlotte-Bristol) may be connected with the Burlington focus.

Chart No. VIII shows the relation in time between the Burlington outbreak and another group of cases in six rural communities clustered together, fifteen to twenty-five miles to the south of that city. The two weeks' interval elapsing between the beginning and the culmination of the epidemic in each of these centers of infection is graphically shown in these charts. In each instance the facts are at least suggestive. This Charlotte-Bristol group may be taken as a typical focus, representative of various other foci in Franklin, Addison, Washington and Lamoille Counties.

While, therefore, we may fairly suspect that the Burlington infection came from Barton, we must also acknowledge



Vermont Poliomyelitis
1914
State Board of Health

Chart No. I

that it is quite possible that the two centers are entirely independent of each other.

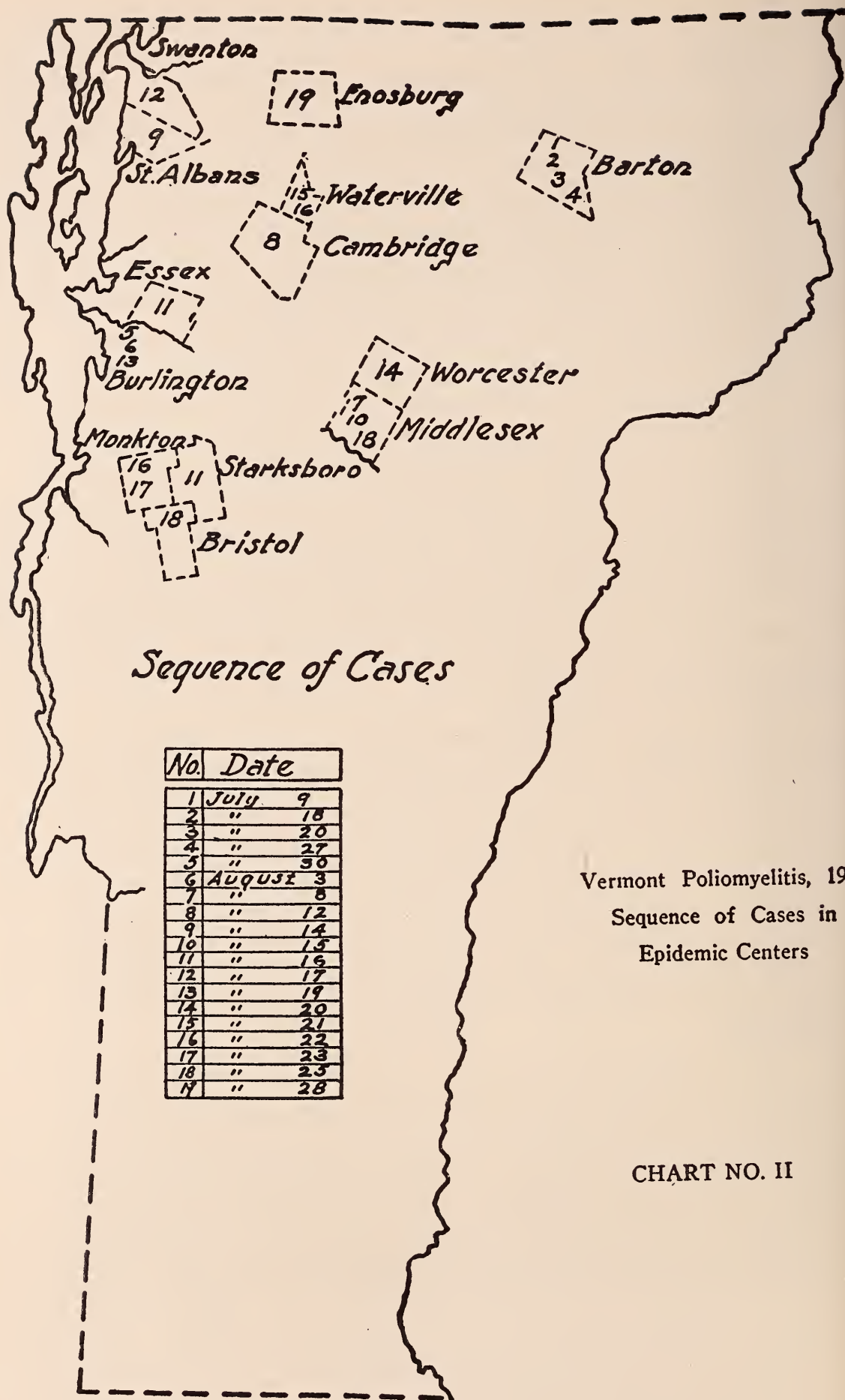
That there were other centers of this infection somewhere in northern New England, New York or Canada, is entirely conceivable; in which event, both of these foci may have been secondary to those in neighboring states. Against this hypothesis in the Barton case is the apparent connection there between the 1913 and 1914 outbreaks.

Both the chronological sequence of cases (II) and the chart (VIII) representing the relation between Burlington and the Charlotte-Bristol group tend to connect the cases which occurred after the middle of August in Franklin, Addison, Washington and Lamoille Counties like those in the Charlotte-Bristol group with the Burlington center.

The presence in Burlington in July and August of circus performances and a merchants' carnival, naturally drawing on all the counties mentioned for patronage, would tend to confirm this supposition.

It has been noted elsewhere in this country, and especially in Sweden, that towns, which have had epidemics of infantile paralysis, are thereafter largely exempt from the disease for varying lengths of time. Wernstedt says, describing the great Swedish epidemic of 1911: "Some of the districts which were severely affected in 1905, were, during the epidemic of 1911 almost entirely encircled by cases of infantile paralysis. But notwithstanding this, they have themselves been left almost untouched by this later epidemic."

The mysterious exemptions during 1914 in Vermont were such towns as Lyndon, St. Johnsbury, Hardwick, Morristown and other railroad towns, which would naturally be on the route of a human carrier traveling between Barton and Burlington. Hardwick, the center of the severe outbreak of 1913, though cases have occurred in adjoining towns, has since been entirely exempt from the disease. Bar-



Vermont Poliomyelitis, 1914
Sequence of Cases in
Epidemic Centers

CHART NO. II

ton and the adjoining town of Irasburg suffered an outbreak in 1910. In that year 8 cases occurred in Barton. At the end of the 1913 outbreak in this section of the state, as has been stated, Barton again had two cases. A four-year interval separated the two severe Barton epidemics. It will be interesting to note if Hardwick has a recurrence of the disease in 1917.

Utterly inexplicable, too, is the fact that many small, sparsely settled rural townships off from main thoroughfares, like Waterville, Washington, Starksboro, Monkton and many others were severely visited. Many times the first cases in a town occurred in families whose members had not been away from home for several weeks and who had received no suspicious visitors. These are facts, of course, that have been widely commented on and can now only be explained as the result of contact with those "agents of dissemination," the virus carriers.

Of the towns in the state which suffered most severely per capita of population this year, the little town of Waterville in Lamoille County heads the list. This town, with six cases, had a case rate of 12.4 per 1000 of population. Other towns followed in this order:

Barton	37 cases or	11.5 per 1000 of population
Washington	7 cases or	9.2 per 1000 of population
New Haven	8 cases or	6.8 per 1000 of population
Bristol	12 cases or	5.9 per 1000 of population
Starksboro	5 cases or	5.9 per 1000 of population
Monkton	4 cases or	5.4 per 1000 of population
Hinesburg	5 cases or	4.7 per 1000 of population
Charlotte	5 cases or	4.2 per 1000 of population
Alburgh	5 cases or	3.8 per 1000 of population
Enosburg	5 cases or	2.2 per 1000 of population
Richford	6 cases or	2.0 per 1000 of population
Burlington	32 cases or	1.5 per 1000 of population

The greatest per capita incidence of the disease by counties was in Orleans County, followed by Grand Isle, Addison, Chittenden, Lamoille and Franklin.

Burlington, with 32 cases and a population of a little over

twenty thousand, did not really suffer so severely from the disease as many other small towns. It was without doubt a prolific center for the distribution of the infection.

CLIMATOLOGICAL

Temperature

The temperature in Vermont during 1914 was not quite up to normal. The chart (No. III) gives the relation of the mean temperature for each month in the year 1914 to the normal for thirty-three years in the state. From this, it will be seen that June, July, August and September were rather below the normal temperature.

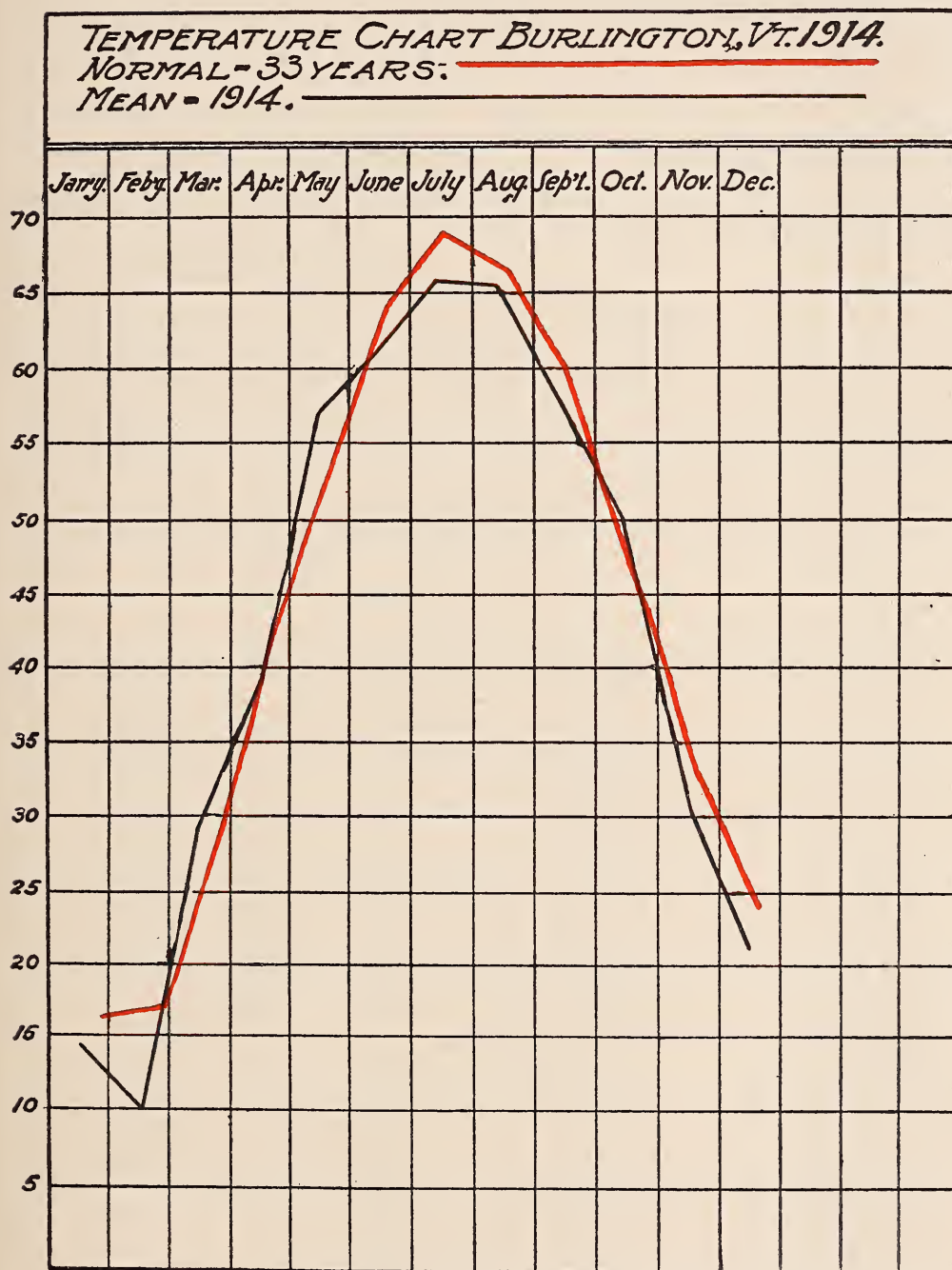
Rain Fall

A deficiency greater or less in rain fall has hitherto coincided in this state with outbreaks of infantile paralysis. Different sections of the state vary widely in the amount of rain fall. The accompanying chart (No. IV) drawn for the purpose of showing the rain fall departures in the state *as a whole* for eleven years, during the summer and fall months, gives the average of observations made at five stations in the state, two of these government weather bureau stations at Burlington and Northfield; the other three volunteer observers at St. Johnsbury, Enosburg Falls and Wells.

The zero line represents the normal with minus averages above this line and plus averages below. The lines representing the departures from normal are the broken lines and give the months for 1914 complete up to November. This chart is an attempt to show graphically the *dryness* of the state, comparing this with the number of cases of infantile paralysis.

The solid lines represent infantile paralysis outbreaks during the last six years. The year 1914, that in which occurred the extensive outbreak we are now considering, was

No. III.



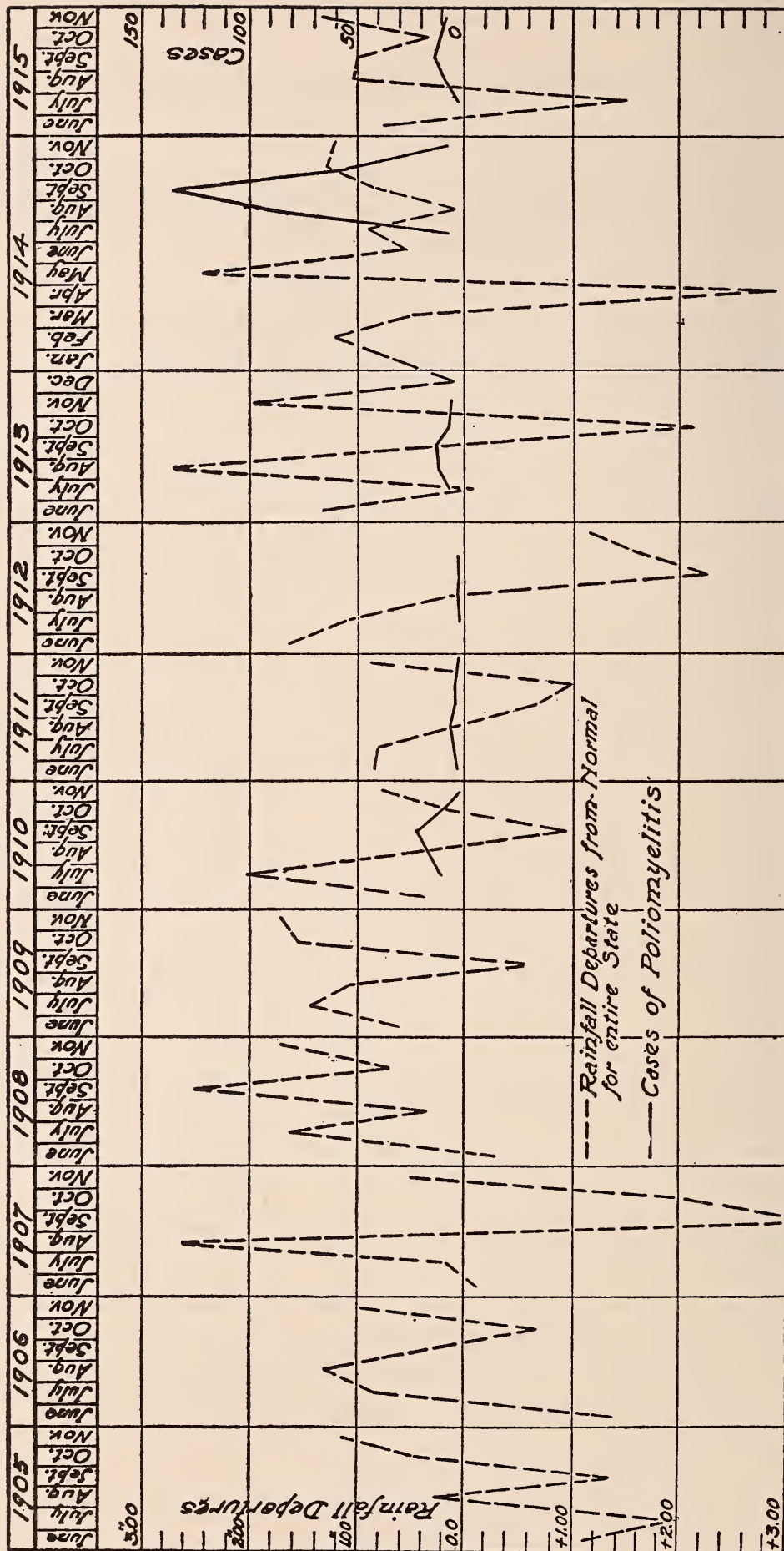


CHART NO. IV

an exceedingly dry summer. The April rain fall was very heavy; thereafter, the balance of the year, exceedingly dry. The deficiency as a whole for that year at Burlington was 9.94 inches. The summer of 1914 was apparently a very dry summer in the state as a whole. The summer of 1908, as shown on this chart, was even dryer, but there was no epidemic that year. Something was lacking to start an outbreak. Nineteen hundred and ten was dry in July and August, but a wet September.

This chart, as mentioned, must fairly show the rain fall of the State of Vermont, as a whole, during the eleven years it covers. The following table gives the temperature and rain fall figures from the Northfield Station, a station which more nearly represents Vermont conditions, as a whole, than any other single station.

	<i>Temperature</i>	<i>Rain Fall</i>	<i>No. Reported Cases</i>
1890.....	40.4	38.17	
1891.....	42.6	31.11	
1892.....	41.3	32.57	
1893.....	39.5	31.36	
1894.....	42.6	28.92	132
1895.....	41.6	35.20	
1896.....	40.8	33.82	
1897.....	41.4	39.14	
1898.....	42.9	30.52	
1899.....	41.4	27.36	
1900.....	41.6	34.11	
1901.....	41.6	31.42	
1902.....	41.2	38.33	
1903.....	41.2	29.09	
1904.....	38.1	27.66	
1905.....	40.0	32.31	
1906.....	40.7	34.75	
1907.....	39.6	37.77	
1908.....	41.4	29.07	
1909.....	40.8	31.98	
1910.....	41.2	31.71	69
1911.....	41.5	27.92	27
1912.....	40.0	37.07	13
1913.....	43.4	31.35	47
1914.....	39.3	30.08	306
1915.....	42.4	28.95	44

(Normal rain fall for 33 years 31.56
 Normal temperature for 33 years 43.00)

Burlington figures, running back to 1828, show that the two lowest records of rain fall since that date were in the years 1894 and 1914 and were 22.62 and 22.96 respectively. Those two years were years of the greatest outbreaks of infantile paralysis which we have had. The Northfield figures given above show that 1894, 1899, 1903, 1904, 1908, 1911 and 1915 were the driest years. The temperature figures seem to indicate nothing.

From these discordant figures, it is perhaps impossible to make any positive deductions as to the relation of rain fall to epidemic poliomyelitis. "Seasonal dryness," however, is a quite regular field observation in connection with this disease with its accompanying low water, dust and insects. The official figures, it must be said, fairly uniformly substantiate this observation.

OUTBREAK IN BARTON

As has been noted, the epidemic of 1914 started in the village of Barton and this village suffered more severely in most ways than any other in the state. The town of Barton is made up of several small villages and the usual farming districts about these. Two of the villages, Orleans and Barton, are incorporated and on the railroad. Of the 37 cases that are reported from this township, 23 occurred in Barton Village; 3 in the Village of Orleans, and the other 11 in the farming districts of the town.

Barton Village is a small manufacturing community of 1330 inhabitants (1910). It is situated on Crystal Lake, whence the Barton River runs through the village. The village has a good water supply from a mountain pond and a fairly good sewerage system. Crystal Lake is a body of water three miles long and about half a mile wide.

The principal industries of the town are wood-working shops, a foundry, three granite sheds, two grist mills and a garment factory.

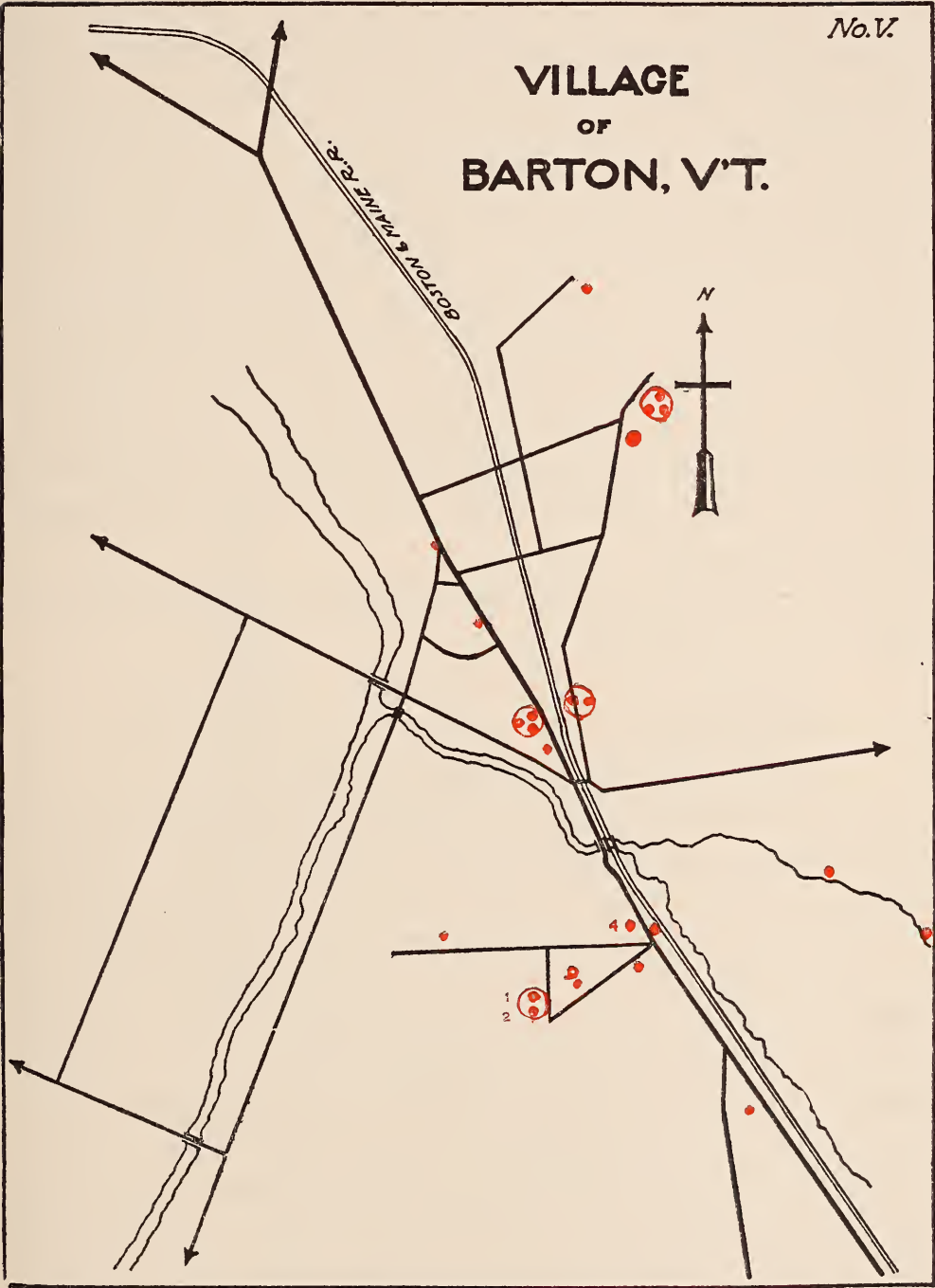


CHART NO. V

As has been noted, the first case recorded in the state this year occurred near the railroad station in this village and rather near to the granite sheds and grist mills. In previous reports of this Board, the possible connection between this disease and stone-cutting industries has been noted, from the severe outbreaks that have occurred in some other stone-cutting centers of the state. This may be, of course, a mere coincidence, as marble and granite industries are the chief industries of Vermont. It should be noted that the earliest cases in this outbreak were in the families of stone-cutters and all the earlier cases seemed to be directly connected by residence or employment with that section of the village near the railroad, which also contained the stone-cutting industries.

The occupations of the bread winners in this Barton outbreak were as follows:

Laborers	6
Stone-cutters	4
Granite-workers	2
Sundry other occupations (one each)	11

It was found that several of the rural cases in the township of Barton very possibly had been in contact with carriers in Barton Village. A fair held in this village between the 15th and 20th of August gave a good opportunity for the distribution of the infection among visitors from the neighboring counties. It should be noted, however, that the epidemic had then reached its height and declined in Barton and the surrounding towns from that time; so that the effect of this fair on its spread is open to question.

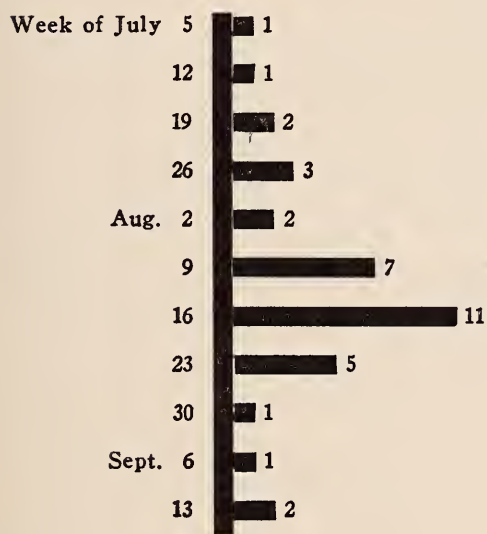
There were several instances of two or more cases in the same house in Barton, as will be seen by reference to the map of that village herewith produced (No. V). The dots enclosed in circles represent two and three cases in those houses. There was unmistakeable connection between several of the frank cases and other frank cases, as well as supposed abortives.

The disease started in July and the last case occurred before the middle of September. The greatest number of cases in the whole town occurred during the week of August 16th and after the week of August 23rd there were only four cases distributed over three weeks.

The schools of the town were not in session and none of the children affected had been in school for more than two weeks before they were taken sick.

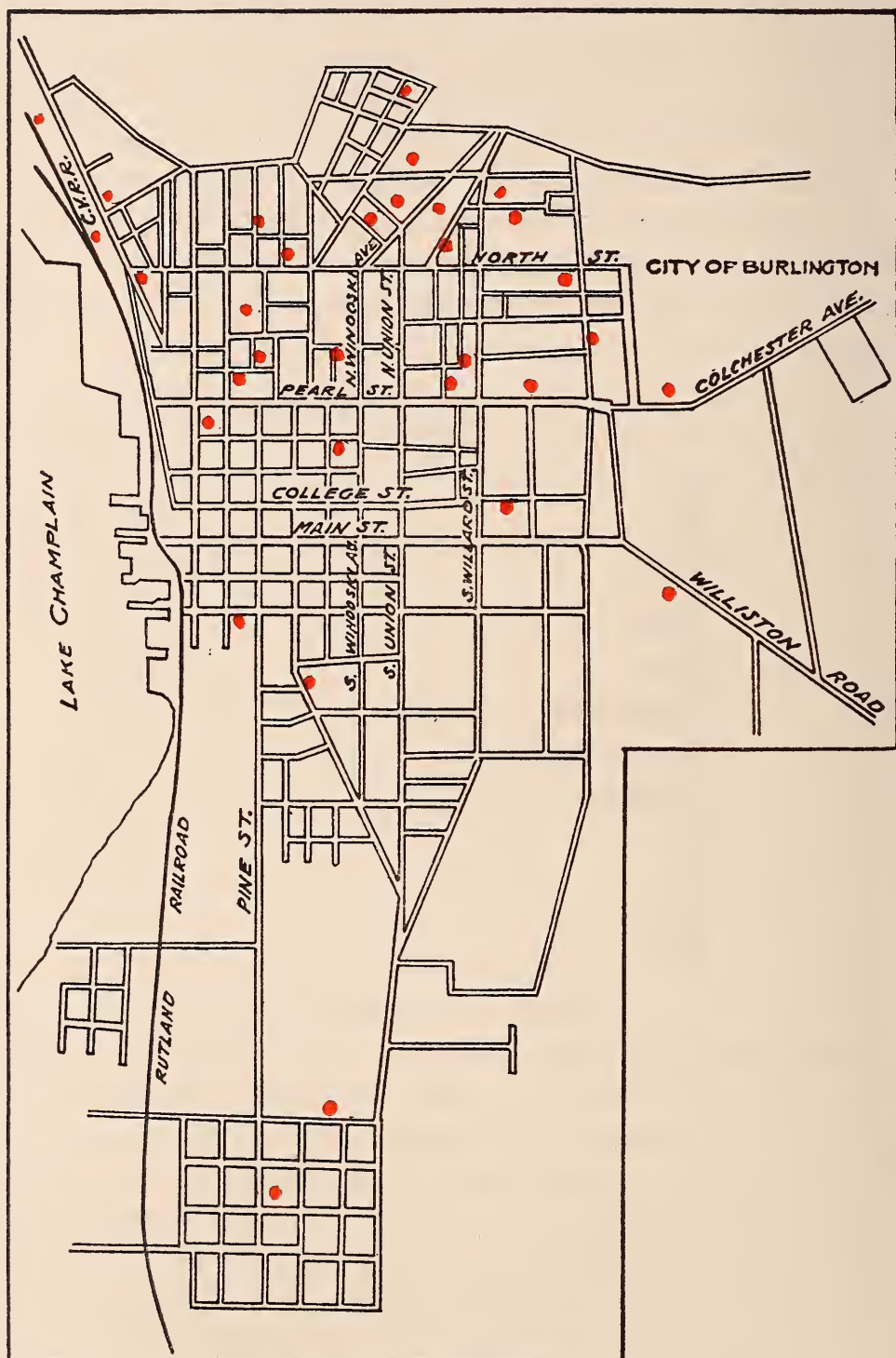
Of the 23 cases in this village 4 died, a mortality of 17.4 per cent.

SEASONAL DISTRIBUTION OF CASES, BARTON (TOWN) OUTBREAK, 1914



BURLINGTON OUTBREAK

The city of Burlington has a population of 20,468 (1910). The portion of the city located on the lake shore is chiefly given up to manufacturing, lumber and railroad yards. Although there are some very unsanitary residences in the southwestern section and on the northern outskirts, the greater part of the area of the city is occupied by well-located and spacious private residences and grounds. The extreme northern section has very small and crowded tenements, often very unsanitary.



BURLINGTON, VERMONT. POLIOMYELITIS — 1914.

VERMONT STATE BOARD OF HEALTH

Aside from the lumber industries, there is a great assortment of minor manufacturing industries in the city, such as stone-cutting, cotton mills, and wood-working shops.

The University occupies a large area at a considerable distance above and from the lake shore. Here, the dwellings and residences occupy spacious grounds and it may be stated with much emphasis that the city, as a whole, is as clean and sanitary as it is beautiful. There is little of the slum aspect to be seen anywhere.

The city water supply is filtered lake water, taken from a considerable distance in the broad lake and the sewerage system finds an outlet on the lake front inside the break water.

The accompanying map shows the location of each case that occurred in Burlington. Three-fourths of the cases occurred in the district north of Pearl Street. While this, perhaps, is not the most sanitary portion of the city, neither is it the most unsanitary, and most of the cases in this section *did not* occur in the worst portion of this section.

The first case occurred on July 30 and most of the others followed during the month of August and the first half of September. The outbreak was rather evenly distributed over seven weeks.

The first case reported was a boy, who was taken sick at a lake resort in the town of Charlotte, whose home was in Burlington. He had been at the lake from the 8th to the 30th of July, when he became sick. Two weeks before coming down, however, he attended a circus in Burlington.

In this connection, as has been mentioned, circuses were in Burlington on July 15 and August 21 and a week's carnival occurred in the city from the 3rd to the 8th of August. There can be little doubt that these large public gatherings, drawing as they did on the surrounding country and especially Franklin, Washington and Addison Counties, may

POLIOMYELITIS-VERMONT, 1914. No. VII.

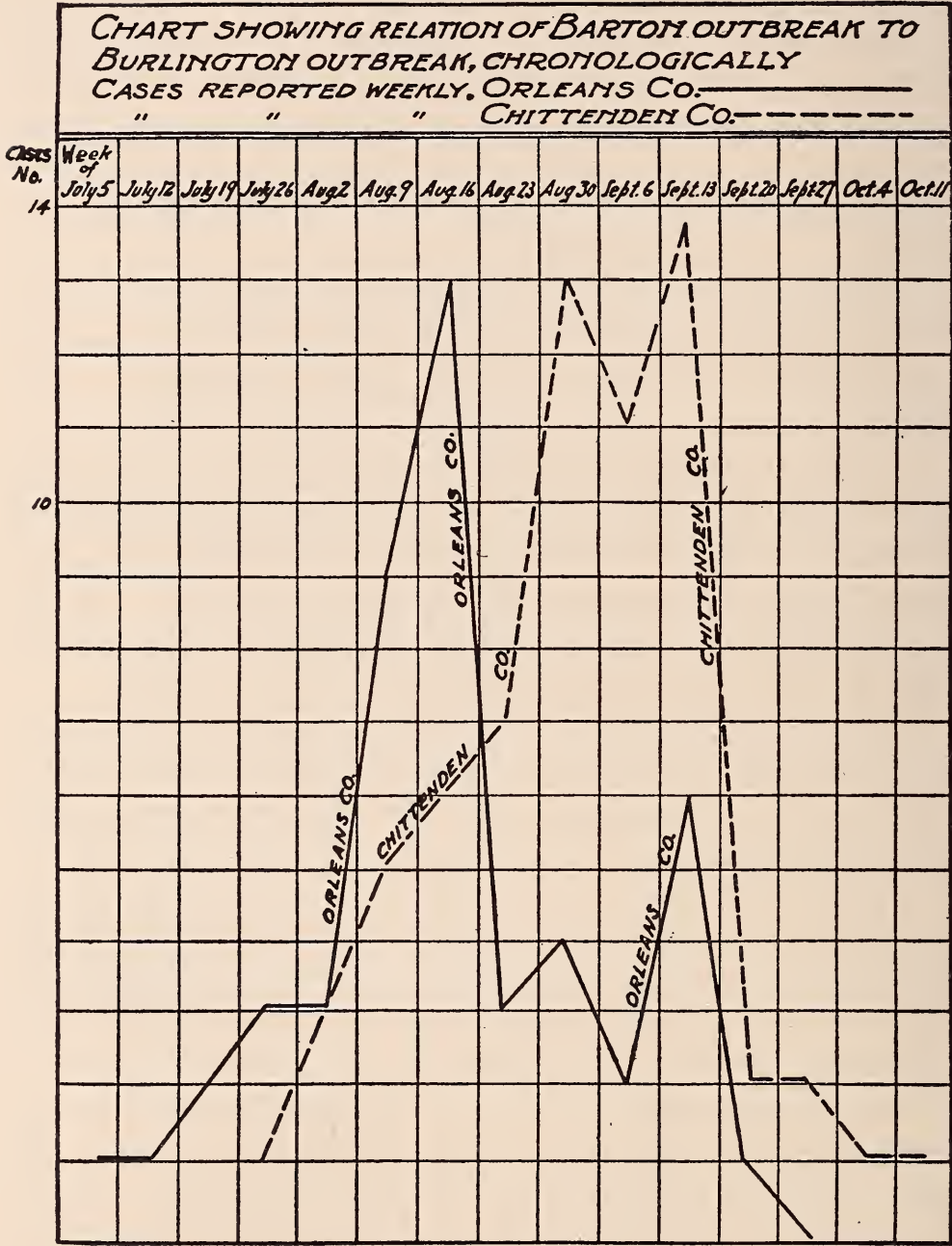


CHART NO. VII

have been a decided factor in the spread of this disease, not only in Burlington, but in the counties mentioned.

As has been noted, this first case was the 5th in the state and the next case in Burlington, coming down on August 3, was the 6th case in the state. Neither of these cases, nor in fact any of the subsequent cases in Burlington, could be traced directly or indirectly to Barton or Orleans County.

Chart No. VII shows the chronological relation of Orleans and Chittenden County outbreaks and is interesting for this reason: the Chittenden County outbreak occurred subsequently to the Orleans County outbreak and at a proper interval of time to arouse suspicion as to their connection. As has been said, other connections cannot be traced. The Orleans County outbreak culminated at the middle of August, and that in Chittenden County, two and four weeks later, respectively, showed two culminating points.

That Burlington furnished the infection for many other communities in neighboring counties is also extremely likely.

The chart No. VIII, which shows the relation between the Burlington outbreak and the collection of cases that occurred in the contiguous rural communities of Bristol, New Haven, Monkton, Charlotte, Starksboro, and Hinesburg chronologically seems to point quite straight to Burlington as the origin of the infection in these towns. The disease began in this cluster of towns two weeks after the Burlington outbreak and had two points of culmination, each two weeks later than the highest point reached by the epidemic in Burlington. Each outbreak existed about the same length of time.

The chart shows that the Burlington outbreak was rather evenly distributed over 6 weeks while the Charlotte-Bristol group of cases had two culminating points: one August 30, and the second one two weeks later. The general identity of

POLIOMYELITIS-VERMONT, 1914. No. VIII.

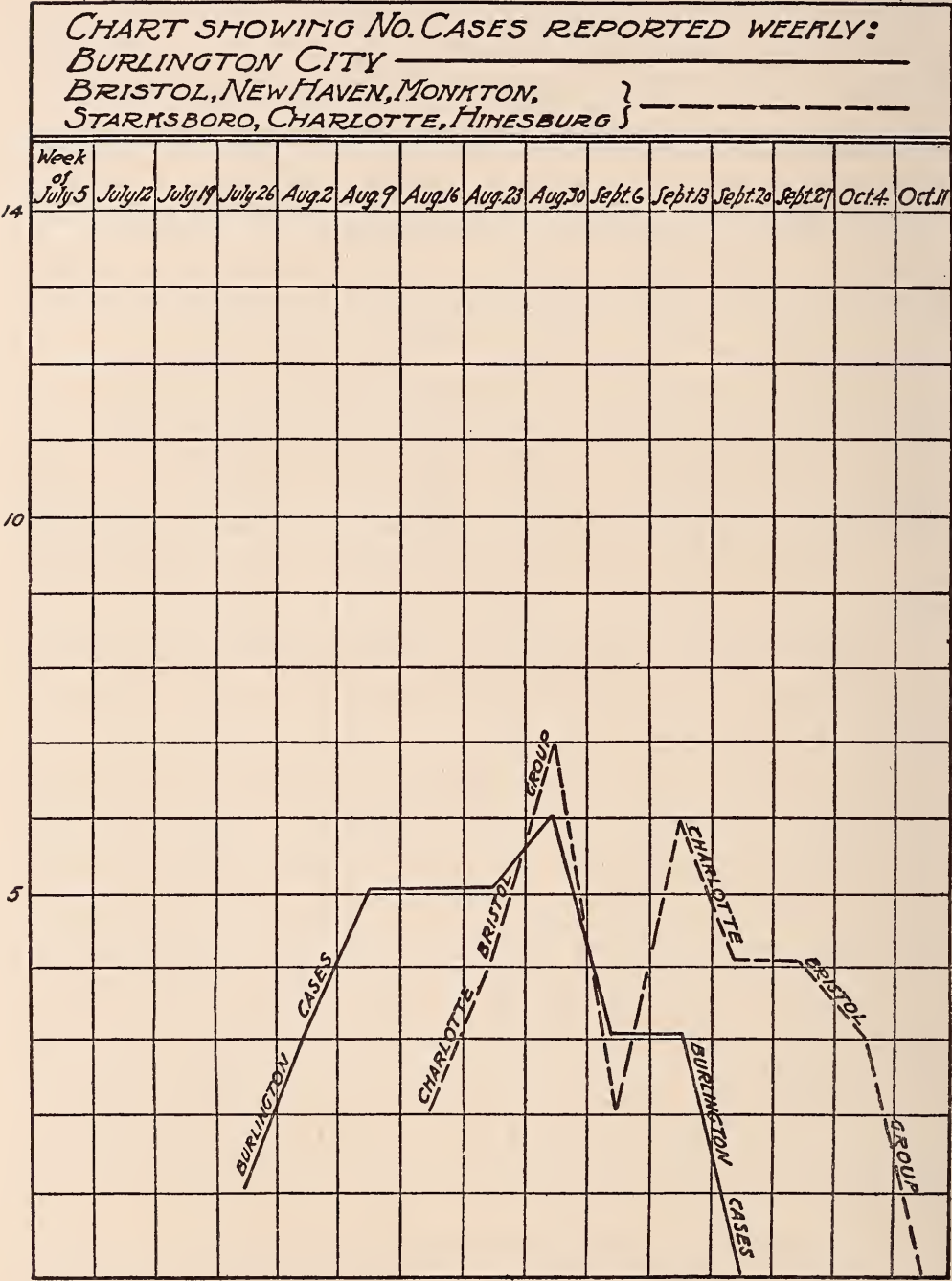


CHART NO. VIII .

the curves, however, with the two weeks separating them is probably more than a coincidence. Two-week intervals seem to separate the crests of local waves of the infection, as note the two charts VII and VIII. Chittenden County and the Charlotte-Bristol group have two separated by that interval.

The Burlington schools were closed at the time of this outbreak, of course, and none of the children had been in the public or parochial schools for fully a month before. Both the public schools and the University delayed opening in the fall three weeks on account of the outbreak. There are, however, two orphanages in the city which have, together, about 280 inmates. One of these in which the greater number is was in the section north of Pearl Street, where the force of the epidemic was chiefly felt. No cases occurred in either of these institutions.

FORT ETHAN ALLEN

In the Town of Essex, six miles from Burlington, and connected by trolley as well as by steam and highway routes, is Fort Ethan Allen, a cavalry post. During the latter part of August the disease attacked a child on this reservation and during the next three weeks six children had the disease, of whom three died. It is known that most of these children were exposed to each other. It might be said, too, that these cases occurred simultaneously with five other cases which occurred in the Town of Essex, in which is the fort. They were, evidently, cases of contact, if at all, with something farther away than the surrounding district of the town. The presumption is that these, like the Charlotte-Bristol group, may be with the other cases in Essex traceable to the city of Burlington. All of these cases occurred under first-class, general, sanitary conditions. They were all in families of officers.

PHILANTHROPY

Not only will the year of 1914 be memorable in the public health records of the state because of the widespread and very serious outbreak of infantile paralysis which visited it, but because this dire event developed a form of practical philanthropy hitherto unknown in Vermont. An anonymous friend of the state placed at the disposal of the State Board of Health a considerable sum of money for the purpose of doing independent and original research work into the nature of this baffling disease, infantile paralysis, and also for the purpose of placing the benefits of expert treatment within the reach of all those who had been maimed by previous epidemics. When this generous benefaction came to us, the Board at once called on Dr. Simon Flexner of the Rockefeller Institute of Medical Research, in New York City, for advice as to the best methods of using this gift. Three distinct lines of work were suggested and have since been carried out. First: an educational campaign for the purpose of giving the medical profession in the state the latest knowledge in regard to the diagnosis and prophylaxis of this disease, especially emphasizing the importance of abortive and carrier cases in its epidemiology. In pursuing this campaign the Board had the services of Dr. Francis R. Fraser of the Presbyterian Hospital in New York City, as well as of Drs. Amoss and Lovett, who were connected with other features of the work. Meetings were held at five different points in the state and everything possible was done to arouse the profession to the importance of this disease and give them the best advice available. The second feature of the work undertaken was original research work into the nature of the infection, its methods of distribution, diagnosis, immunity, etc. This work has been in charge of Dr. Harold L. Amoss of the Rockefeller Institute in New York. A Research Laboratory was established in the College of Medicine, the University of Vermont, where Dr.

Edward Taylor is working under Dr. Amoss' direction. Reference is hereby made to the report of this department. The third and last line of work undertaken by the Board under this benefaction was the treatment of paralyzed cases. Dr. Robert W. Lovett, Chief of the Orthopedic Department of the Children's Hospital in Boston, has had full charge of this work. Again reference must be made to his report for details of the valuable work done and results obtained.* Not only have these Vermont cases been most surprisingly benefited, but with Dr. E. G. Martin of the Physiological Department of Harvard University, Dr. Lovett has evolved a method of measuring the strength of muscles, sound and impaired, which is likely to have far-reaching results in the management of this disease.

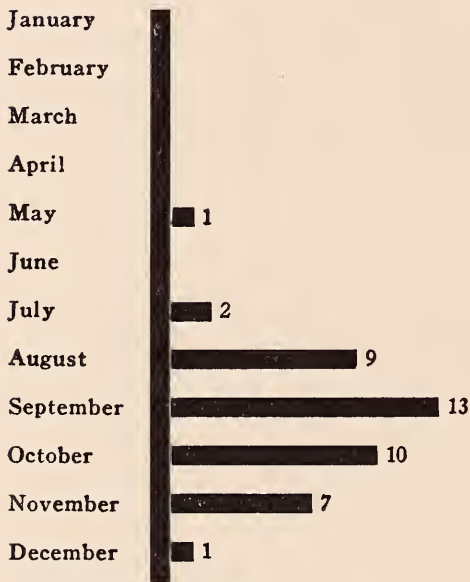
1915

In 1915, 44 known cases of infantile paralysis occurred in the state. After the very serious outbreak of 1914 and owing to the campaign instituted by the State Board of Health under its Special Fund, as mentioned above, the physicians of the state, as well as most of the citizens, were alert to the possibility of a recurrence of this disease in epidemic form. There was a general feeling in the profession that the disease would not attack the northern part of the state, but might the southern this year. The sequel shows that we were again mistaken. The profession, generally, were so alert to the possibility of outbreaks of this disease that the State Board of Health and the experts employed by it were in demand during the summer months for the purpose of investigating suspicious cases. Dr. Taylor, in charge of the research laboratory, personally investigated many of these cases. Physical examinations and lumbar punctures were made in almost all for the purpose

*Journal A. M. A. Mar. 4, 1916. P. 729.
Vermont Medicine. Feb'y, 1916. P. 36.

of diagnosis, and many autopsies were made by Drs. B. H. Stone and Taylor. It might be said in this connection that the number of cases of tubercular meningitis that developed from these investigations was rather surprising. Many suspicious poliomyelitis cases proved to be that disease. The same blanks were used in collecting data in 1915 as were used in 1914.

1915
POLIOMYELITIS—VERMONT
*Seasonal Distribution**



*One case not stated.

It will be noticed in the above chart that the disease, as usual, occurred during the latter part of the summer and fall, culminating in September.

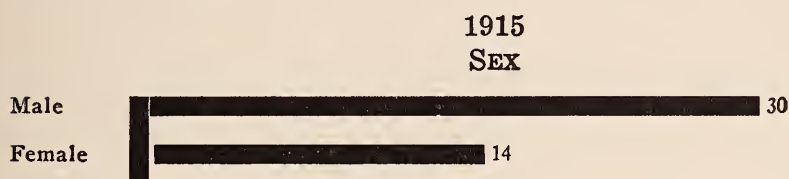
The preponderance of male cases here shown is much greater than usual.

Last year the Vermont cases were divided 55 per cent male to 45 per cent female.

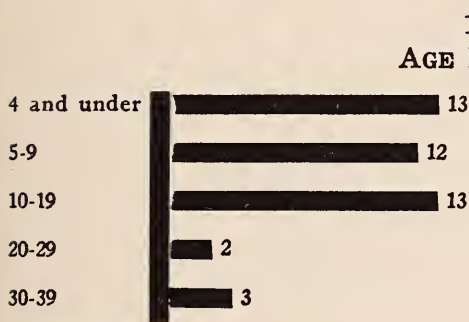
The 1915 Vermont cases numbered 68.2 per cent males and 31.8 per cent females.

The age distribution in these cases is shown in the fol-

lowing chart. Perhaps the only comment that is called for in this connection is the proportionately large number of cases between 10 and 19 years.



The combined sex and age distribution according to counties is given in the following table:



*One case age not stated.

1915 SEX AND AGE DISTRIBUTION											
	4 & under		5-9		10-19		20-29		30-39		Total
	*M	F	M	F	M	F	M	F	M	F	
Addison			1	0							1
Bennington			9	1							1
Caledonia	2	0	3	2	4	2			0	2	15
Chittenden	1	1	0	1	0	1					4
Essex							1	0			1
Franklin					1	0					1
Grand Isle	1	9									1
Lamoille											
Orange					1	0					1
Orleans	2	0	2	1	1	1	1	0			8
Rutland	1	0			2	0					3
Washington	0	1									1
Windham									1	0	1
Windsor	3	1	1	0							5
Not stated											1
Total	10	3	7	5	9	4	2	0	1	2	44

*M Male; F Female.

While the majority of the cases occurred in "villages,"

these villages are all distinctly rural. They are usually the typical New England villages, with small population ranging from, perhaps, 500 to 1,000 people. The essentially rural portions of the state were again rather severely visited. Indeed, the apparent preference of this disease to rural life is conspicuous in the distribution of the disease this year. The nationality, occupation of wage earners, and general sanitary conditions are shown in the following tables:

1915
NATIONALITY

<i>Father</i>		<i>Mother</i>	
American	28	American	29
Canadian	9	Canadian	9
English	1	English	1
German	1	German	0
Austrian	1	Austrian	0
Italian	0	Italian	1
Polish	2	Polish	2
Irish	1	Irish	1
Not stated	1	Not stated	1
Total	44	Total	44

1915
OCCUPATIONS

Farmer	12
Laborer	9
Railroad section hand	2
Traveling man	2
Sundry other occupations (one each)	19
Total	44

1915
CONDITION OF PREMISES

Excellent	8
Good	15
Fair	13
Bad	8
Total	44

1915
STATUS OF FAMILY

Well-to-do	7
Moderate	20
Poor	17
Total	44

1915	
ONSET OF PARALYSIS	
1st day	1
2nd day	11
3rd day	8
4th day	10
5th day	3
6th day	1
7th day	0
Later than 7th day	3
Not stated or not paralyzed	7
Total	<hr/> 44

DISTRIBUTION OF PARALYSIS

As in 1914, a great variety of combinations were obtained in the muscles and groups of muscles affected in this milder outbreak of 1915. More delicate and precise methods of muscle-testing must continually bring to light more and more bewildering combinations of paralyses in these cases of infantile paralysis. The commonest paralyses noted in this outbreak, including the motor paralysis in several fatal cases, were:

All extremities	3
Left arm alone	1
Left arm and left leg	1
Right arm alone	1
Right arm and both legs	1
Right arm and left leg	1
Both legs alone	3
Both legs and certain trunk muscles	4
Left leg alone	5
Right leg alone	5
Right leg and certain trunk muscles	2

Aside from these 27 cases, there were, as usual, a number of cases in which the chief paralysis was hard to locate or in which it was vaguely stated. Such were "facial," "one leg," "neck muscles," "deglutition," "arm, neck and face."

Results

The end results in this series of cases are given in the following table:

Paralyzed	24
Died	11
Recovered (abortive)	9
Total	<hr/> 44

Two facts are noticeable in the above table. The high mortality (25 per cent) is the first. The mortality in this disease of late years has run somewhere between 10 and 17 per cent. The mortality of 17.3 in this state in 1914 in the large series of cases was very high. These figures for 1915 are rather disquieting. It was believed by observers that the disease was really of a more malignant type, especially in certain towns. A second fact brought out by the above table is the large proportion of recovered (abortive) cases. This is easily explained. The attention of physicians had been so generally directed to the possibilities of a recurrence of this disease in the state this year, that suspicious cases were very carefully investigated. It goes without saying that some of these cases which would formerly have passed for "colds," "grip," "indigestion" or "teething" were occasionally found to be really poliomyelitis. While our methods of making diagnoses in these cases are still very imperfect and unreliable, only such cases have been included in this list as there were good reasons for thinking belonged there. Some such reasons were: the occurrence of cases in conjunction with the frankly paralyzed children, the typical initial symptoms followed by prolonged invalidism, and finally the occasional detection of weak muscles by means of the methods devised by Drs. Lovett and Martin.

FREQUENCY OF ABORTIVE CASES

The frequency with which abortive cases occur in outbreaks of this disease is still uncertain. In the 1914 epidemic of 306 cases, 26 were so classified. In outbreaks reported elsewhere estimates of the number of abortive

cases have varied widely. The ratio of paralyzed to abortive cases in Massachusetts in 1909 was reported as 3 to 1. In the same state in 1910, 4 to 1. In Iowa, 1910, the abortives were placed at 19 per cent of all. In Sweden the percentage of abortives has been estimated in various outbreaks all the way from 15 per cent to 56 per cent. Careful observers would now place the proportion in most epidemics higher than formerly. For these reasons, the number of cases (26) classed as abortive in our 1914 epidemic was probably far under the truth. Our 1915 experience is more in keeping with the recent observations of this disease.

Four brothers in a family in an isolated part of Wheelock came down with the disease; the first on October 22, the others following on October 31, November 1, and November 2. One of these cases was fatal and one abortive. One case (Irasburg) occurred in a person who was said to have had the same disease 18 years before. There was one instance in which a clear connection was traced between two cases seven days apart and still another in which a paralyzed case was in contact with an abortive seven days previously.

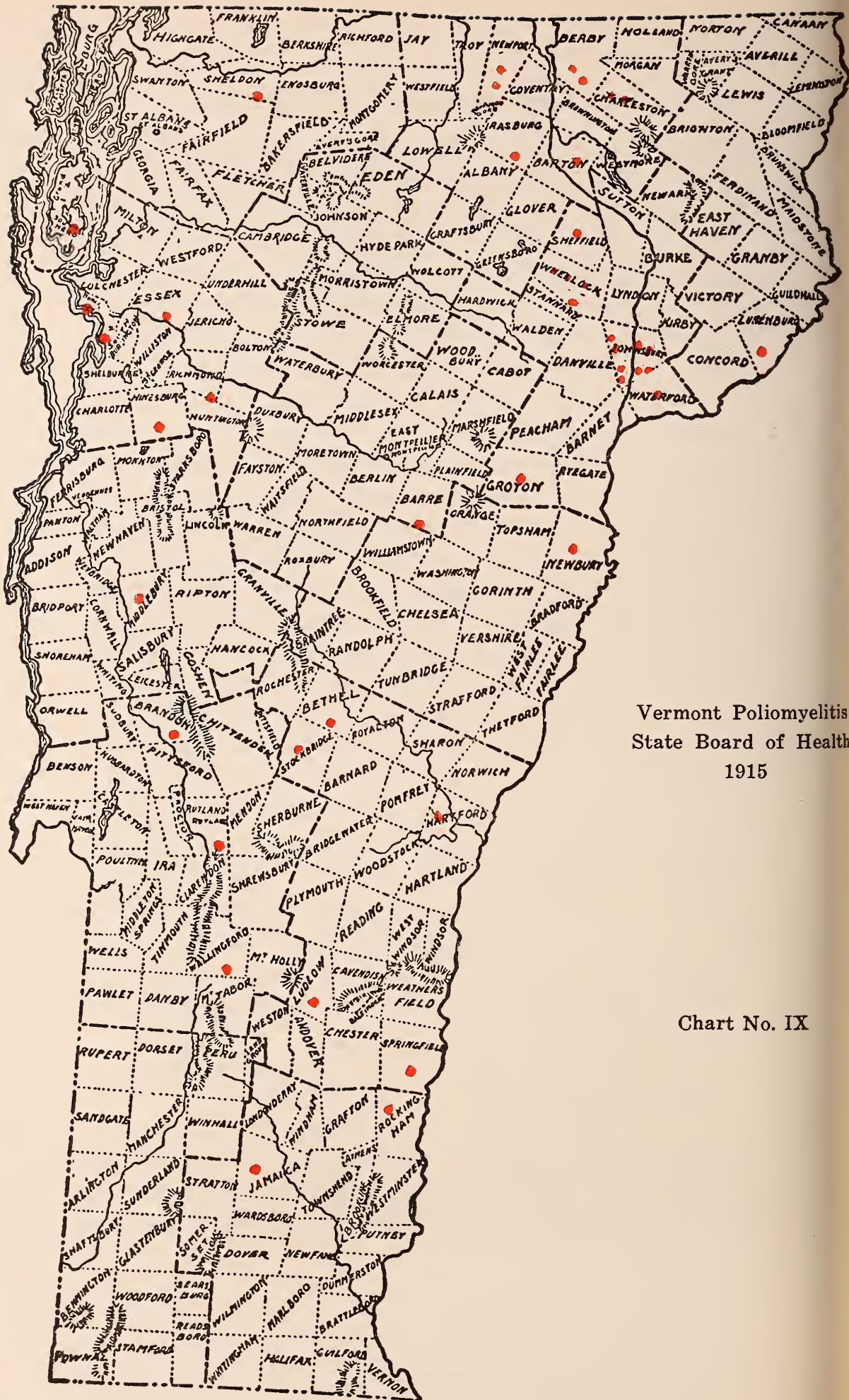
The death rate by age periods in 1915 was as follows:

PERCENTAGE OF DEATHS

By Age Periods

Of 13 cases, 4 years or under	30.7% died
Of 12 cases, 5-9 years	16.6% died
Of 13 cases, 10-19 years	23.0% died
Of 2 cases, 20-29 years	No deaths
Of 3 cases, 30-39 years	66.6% died
Age not stated in one case.	
Of all cases 25.0% died.	

References to the Map No. IX will show that the cases in 1915 were quite generally distributed over the state. The only section suggestive of epidemic conditions is in the val-



Vermont Poliomyelitis
State Board of Health
1915

Chart No. IX

leys of the Passumpsic and Barton Rivers. One-half of all the cases occurring in the state this year were in these valleys. This suggests an aftermath of 1914.

1915

SEASONAL AND GEOGRAPHICAL DISTRIBUTION

East

<i>County</i>	<i>May</i>	<i>June</i>	<i>July</i>	<i>Aug.</i>	<i>Sept.</i>	<i>Oct.</i>	<i>Nov.</i>	<i>Dec.</i>	<i>Total</i>
Caledonia				1	6	5	3		15
Essex					1				1
Orange				1					1
Orleans					2	4	1	1	8
Washington				1					1
Windham						1			1
Windsor			1	1	1		1		4
									<hr/> 31

West

Addison	1								1
Bennington				1					1
Chittenden				2	1		2		5
Franklin				1					1
Grand Isle				1					1
Rutland			1		2				3
Not stated									1
Total	<hr/> 1	<hr/> 0	<hr/> 2	<hr/> 9	<hr/> 13	<hr/> 10	<hr/> 7	<hr/> 1	<hr/> 44

Eight of these cases occurred in September, 9 in October, and 4 in November. The first case in this local area occurred August 26 in St. Johnsbury. Of the remainder of the cases in St. Johnsbury, 6 occurred during the month of September.

The first case in Orleans County this year occurred September 6 in Barton township. This case could not be traced to any possible connection with St. Johnsbury, unless, perchance, some carrier or missed case from St. Johnsbury may have attended the Barton Fair, August 18, which was also attended by this case. It will be remembered that St. Johnsbury has almost entirely escaped cases of this disease until this year. Of the 8 cases occurring this year, 3 were fatal.

Again it may be stated that 1915 was not a hot year, but was dry. Taking the Northfield figures, the mean temperature for the year was 42.4 against a normal of 43. The rain-fall deficiency, however, was marked, viz., 2.61 inches. The coincidence of our outbreaks of infantile paralysis, it may be repeated, with *dry* summer months, is at least significant.

CONCLUSIONS

A review of our experiences with this disease in Vermont warrants the following observations:

The disease seems to be a rural disease. Something in rural life apparently favors its propagation and spread.

The disease, while apparently following the arteries of human intercourse, makes long jumps between towns and attacks persons in isolated and inaccessible regions.

August, September and October are the favorite months for the disease in Vermont.

A community visited by an epidemic of this disease has, apparently, comparative immunity thereafter for several years. The experience of Barton might suggest a four-year period.

As has been stated, poliomyelitis, under our regulations, is reportable and subject to "Full Quarantine" and terminal disinfection. In the present state of our knowledge of its epidemiology, there may be a theoretical question as to the utility of the quarantine. Practically quarantine measures, well carried out, *appear* to check community outbreaks. So in the absence of more positive proof of its inutility, we would not be warranted in abandoning it.

In the presence of an epidemic, public gatherings, like picture shows, public, parochial and Sunday schools, fairs and circus performances, largely frequented by children, should be prohibited.

In this connection, the following from Dr. Flexner's pa-

per summarizes our present knowledge of the ways the infection is spread:

"The data which I have had the pleasure of laying before you have led me to believe, first, that the microbic agent of epidemic poliomyelitis is present in the nasal and buccal secretions and is carried by persons, not insects, and communicated by them in such manner as to gain access to the upper respiratory mucous membranes of other persons, among whom a portion, being susceptible to the injurious action of the virus, acquire the infection and develop the disease.

"The clinical variety or form of the disease which they develop may be the frankly paralytic, the meningitic, or the abortive and ambulatory in which no severe symptoms whatever appear. But however the persons may be affected, they become potential agents of dissemination of the virus of poliomyelitis, as do a number of healthy persons who have been in intimate contact with those who are ill, and another group of persons who have recovered from an acute attack of poliomyelitis. These several classes of infected or contaminated persons constitute the active means through which the virus is spread and to the control of which sanitary measures designed to prevent epidemics must be directed."

Finally, the writer wishes to acknowledge the assistance given by Dr. H. A. Ladd, Sanitary Inspector, in securing field notes of these outbreaks, and by Dr. C. F. Dalton, Secretary, in compiling these statistics. The work of Prof. J. W. Votey, Engineer of the Board, on the climatological data has been indispensable.

To local health officers of towns invaded, especially to Dr. M. R. Prime of Barton, our acknowledgments are due for loyal support in carrying out the regulations of the State Board, and furnishing data for this report.

INFANTILE PARALYSIS (POLIOMYELITIS) IN VERMONT 1916-1917*

By CHARLES S. CAVERLY, Sc.D., M.D.

1916

THERE were 64 cases of this disease recognized in Vermont during the year 1916. The most of these cases, as will be pointed out, belonged probably to the outbreak which invaded New York and adjoining states from the severe epidemic which had its focus in Brooklyn.

The earliest cases in Vermont appeared in Arlington, Pawlet, Poultney and Woodstock during the last of August. One case, in which the diagnosis was somewhat doubtful, had occurred in the town of Underhill on June 28. The real epidemic, however, started in Arlington, August 20. This was a late date for the epidemic occurrence of this disease, even in Vermont, where it has usually occurred rather later than in neighboring states. Four of the eight cases which occurred in Vermont during the last ten days of August were in this town. Three of these cases occurred in one family. Fourteen days previous to the first case, the father of the children had made a two days' trip through parts of Connecticut, Massachusetts and New York by automobile. The second and third cases in this family developed four and six days after the first. The fourth case in the town of Arlington occurred in a family, in which the grandmother of the child had been in contact with the father of the three children previously mentioned, eleven days before this fourth child was taken sick. These are the only facts elic-

*Reprinted from Bulletin of the Vermont State Board of Health, Vol. XIX, No. 1, Sept., 1918.

ited in regard to the origin of these first cases in our 1916 outbreak.

Cases of the disease, widely scattered, occurred in various parts of the state through September and October. Grand Isle in Lake Champlain, a sparsely settled island, had the most pronounced epidemic focus observed. These cases began about September 20.

Because of the repeated experience with this disease during the last seven years, and because of the Special Fund, anonymously given to the State Board of Health for Polio work, we were better equipped for handling an outbreak than ever before. The progress of the disease, after its appearance in New York in June, had been watched with interest here and cases were not unexpected. We had taken what seemed to us proper precautions early in the summer against the spread of this disease from New York City into our state. Infantile paralysis has been reportable in Vermont since 1910 and the Regulations of the State Board of Health require a full quarantine of twenty-eight days. This quarantine includes all persons on the premises. The disease is reported by the medical men of the state, as far as recognized, and the quarantine regulations are strictly observed. Data of this outbreak were collected on blanks similar to those used in Vermont for two years and these were filled out by either the attending physician, or by the Inspector of the State Board, Dr. Ladd, or the special investigator, Dr. Taylor. These data in condensed form follow:

INCIDENCE OF THE DISEASE BY AGE AND SEX—1916

Sex



Of the 64 cases occurring this year, 36 were males and 28 females. Males 56.2 per cent, females 43.8 per cent. This

is approximately the same division, as regards sex, that occurred in the great epidemic in this state in 1914.

Among 1,081 cases studied in New York State this same year, the percentage of males was 53.4; while among the more than 9,000 cases, which occurred in Greater New York City, the males were slightly over 54 per cent.

The following diagram represents the age incidence of the disease in Vermont this year.



The percentage of young children in our Vermont epidemics has been very low as compared with urban outbreaks. The number of children under five years of age in this epidemic was 22. This is 34.3 per cent of the whole number of cases; in 1914, of 304 cases, which occurred in the state, 38.1 per cent were under five. These figures are remarkably low, when compared with the figures for Greater New York in this 1916 epidemic. Of 9,345 cases in that epidemic, 77.3 per cent were under five years of age; and of 1,081 cases in New York State, outside of New York City, the same year, 50 per cent were under five. On the other hand, during our epidemic, 28 per cent of the cases were ten years of age or over; in New York City, the same year, only 3.6 per cent were ten years or over; and in the state, outside of the Greater City, 12 per cent were ten years of age or over. In other words, our percentage of young children to date has been very low, and of youth and adults high.

In the Pennsylvania outbreak of 1910, the percentage of cases under five was 72. In Buffalo, in 1912, 75 per cent of the cases were under five; and in Springfield, Mass., and vicinity, in 1912, the percentage under five was 71. On the other hand, in the Iowa epidemic in 1910, only 24.3 per cent of the cases were under five.

These figures suggest the question whether there may be something in urban life that increases the susceptibility of young children to this disease.

The combined distribution of the cases by age and sex follows:—

	4 & under		5-9		10-19		20-29		30 & over		Total
	M	F	M	F	M	F	M	F	M	F	
Addison			2	0	0	1					3
Bennington	2	1	1	0	4	1	1	0			10
Caledonia	1	0	0	1							2
Chittenden	1	3	0	1	1	1	0	1			8
Essex											0
Franklin			1	0							1
Grand Isle	2	2	4	3	1	1	0	1			14
Lamoille			1	0							1
Orange	2	0	0	1			0	1			4
Orleans					0	1					1
Rutland	4	1	6	3	0	1	0	1			16
Washington					0	1					1
Windham	1	0									1
Windsor	2	0									2
Total	15	7	15	9	6	7	1	4	0	0	64

ONSET OF PARALYSIS

Among 43 of these cases, in which the information was obtainable, the onset of the paralysis was on or before the 4th day, in 74+ per cent.

The following table gives the day on which the paralysis was first noted:—

Same day	3 cases
1st day	4 cases
2nd day	13 cases
3rd day	8 cases
4th day	4 cases

5th day	1 case
6th day	6 cases
7th day	1 case
After 7th day	3 cases

The distribution of the paralysis in these cases, excluding thirteen cases that were "abortive" and showed no paralysis, also one in which it was never determined whether there was any distinct paralysis, and one other in which the diagnosis was made post mortem was as follows:

DISTRIBUTION OF PARALYSIS

All the extremities were wholly or partially paralyzed in ..	1 case
Left arm alone in	2 "
Left arm and both legs in	1 "
Right arm alone in	1 "
Right arm and both legs	1 "
Right arm and right leg	1 "
Facial paralysis alone in	1 "
Both legs alone in	9 "
Left leg alone in	6 "
Right leg alone in	8 "
Respiration	3 "
One leg	1 "
Deltoid muscles and right side	1 "
Legs and face	1 "
Arms, bladder and respiration	1 "
Whole body, except one arm	1 "
Right side	2 "
Left ankle and foot	1 "
Respiration, bowels and bladder	1 "
Legs and respiration	1 "
Both legs and bladder	1 "
Complete paralysis	1 "
Right arm and respiration	1 "
Throat, respiration and arms	2 "

RESULTS

Paralyzed cases who survived numbered	32
Died	12
Fully recovered, including abortive cases	19
Died in another state	1

PERCENTAGE OF DEATHS

By age Periods

Of 22 cases, 4 years and under	18.18% died
Of 24 cases, 5 to 9 years	25.0% died
Of 13 cases, 10 to 19 years	7.69% died
Of 5 cases, 20 to 29 years	20.0% died
Of 64 cases (All ages)	18.75% died

PERCENTAGE OF DEATHS BY SEX

Of 36 male cases	5 died or 13.8%
Of 28 female cases	7 died or 25.0%

The usual mortality in cases of infantile paralysis is greater among the males than the females. The figures are reversed with our cases this year. The mortality this year was about the same as has happened heretofore in Vermont. In 1914 it was 17.3 per cent. In New York City this year the case fatality was 26.9 per cent. In that state the mortality was 23.7 per cent. In Boston this same year the mortality was 32.8 per cent, and in towns of 2,500 or less inhabitants in Massachusetts in 1916 it was 23.4 per cent. The mortality in any epidemic, of course, varies with the thoroughness with which the cases are diagnosed and reported. The more thoroughly the cases are reported, the less the apparent mortality in any epidemic of course varies with the thorough-abortive or mild, as well as paralyzed. In other words, rural poliomyelitis would seem to be of milder type than urban.

As usual with this disease, there were numerous instances in which there were other children, in contact with the sick child in this outbreak, who escaped. The following figures represent such instances:

OTHER CHILDREN UNDER 20 IN FAMILY (PARALYZED CASES)

1 other child under 20 in family	8 instances
2 other children under 20 in family	14 instances
3 other children under 20 in family	6 instances
4 other children under 20 in family	4 instances
5 other children under 20 in family	7 instances

TWO OR MORE CASES IN SAME FAMILY

In families with 2 children under 20—2 instances of more than 1 case
 In families with 3 children under 20—5 instances of more than 1 case
 In family with 4 children under 20—1 instance of more than 1 case
 In family with 5 children under 20—1 instance of more than 1 case

CONTACTS

The more epidemics of infantile paralysis are studied, and especially the pre-paralytic symptoms, on which a diagnosis of the disease may be based, the larger percentage of cases are found which may be reasonably ascribed to *contact*. In this epidemic of 64 cases, 15 were found to have been in contact with a frank paralytic case; three others had been in contact with either abortive or carrier cases, and in 46 instances no such contact could be traced. Thirty-nine per cent, in other words, of these cases, were traceable to possible contact infection; while in the large epidemic of 1914 in this state, only 22 per cent were so traceable.

In 39 families with other children in this outbreak, in which cases occurred, only 9 developed secondary cases. While, therefore, contact infection seems to be increasingly traceable, it must still be considered a disease of rather low contagiousness.

The three cases in the Arlington family before mentioned were the earliest cases that had occurred in the state. The possible connection which those cases had with the infection in one of the neighboring states has also been mentioned. The geographical distribution of the cases in this epidemic and the possible relation to New York cases, which this distribution suggests, will be mentioned later. There were several instances in which either the patient or some other member of the family had either been in contact with paralytic cases or had come from distant towns in this or neighboring states where the disease was prevalent. For instance, an adult female of twenty-three years of age, who had the disease in September in the town of Underhill, had been, for some weeks prior, in daily contact with a probable case sent from a city where there were cases in a neighboring state to recuperate. This probable case had been sick some weeks before and had a weak arm and weak legs as the result. This case, together with three other persons

from the same city, had visited in the family of the person sick in Underhill for two weeks before she was taken sick.

Another instance of possible carrier infection occurred in the town of Brookfield. Three children in the family of A. J. were sick in September. A girl five years old was taken sick September 9. She died in convulsions on the 17th with paralysis of the legs. An autopsy done on this case showed unmistakable polio lesions of the cord. A boy three years old in the same family, taken sick on the 15th, died on the 18th, with paralysis of both arms and respiratory muscles. A baby in the same family, three months old, was taken sick with febrile symptoms and marked rigidity of the neck and spine, September 18. The sister of Mrs. A. J., who lived in Springfield, Mass., visited in Hartford, Conn., on August 27. At the house where she was staying was a child who was diagnosed that day as having poliomyelitis and died the following day, August 28. This woman and her husband immediately left the house in Hartford and returned to Springfield, and on September 3, they went to Brookfield, where they stayed until the 15th with Mr. and Mrs. A. J. The three children were taken sick from the 9th to 18th of September. These instances of possible infection are being more frequently noticed as the disease is being more carefully investigated.

TONSILS AND ADENOIDS

Not all of our cases this year were carefully examined for nasal or throat disease. Our records showed there were eighteen cases in which there were marked symptoms of adenoids or enlarged and diseased tonsils. One case had been operated on for adenoids within three years.

OCCUPATION OF BREAD WINNER

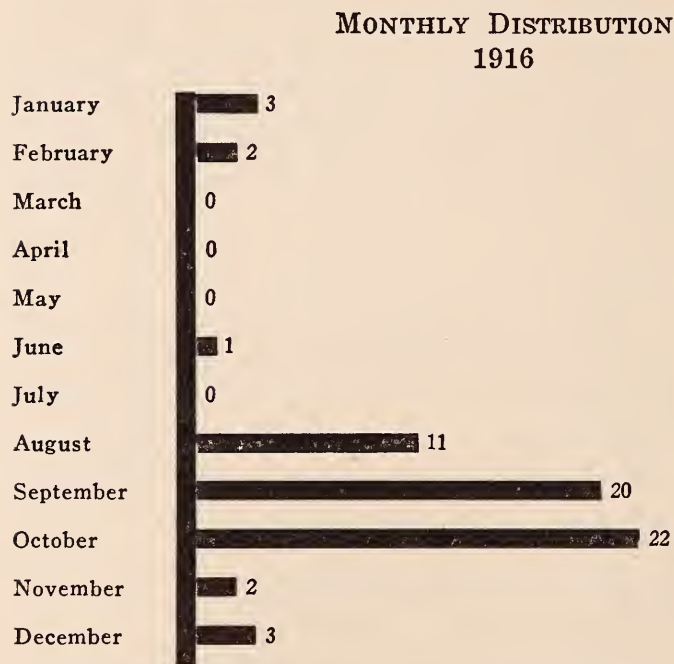
The occupation of the head of the family in these cases was as follows:

Laborer	22
Farmer	20
Teamster	4
Slate quarry man	3
Mechanic	6
Marbleworker	2
Various other pursuits (one each)	7
	<hr/> 64

Other facts in regard to these cases were as follows:—

NATIONALITY 1916			
<i>Father</i>		<i>Mother</i>	
American	36	American	33
French	20	French	23
Canadian	2	English	2
Welsh	2	Welsh	2
Scotch	2	Irish	1
English	1	Canadian	1
Austrian	1	Austrian	1
	<hr/> 64	Spanish-Mexican .	1
			<hr/> 64

The distribution of the cases, by months, is represented on the following chart:—



The real epidemic of the year began in August, attained

its height in October, rapidly dying out with the approach of cold weather.

The disease in New York City began early in June, the cases appearing in great number after the third week. The epidemic reached its maximum in August and declined thereafter gradually until November.

The cases in New York State, outside of the city, began to appear in considerable numbers the first week of July; and those in Washington County, which is contiguous to Bennington and Rutland Counties in Vermont where the first cases of our epidemic appeared, occurred, one case the first week of July, and three or four more during the third and fourth weeks of August. The most of the cases, therefore, occurred at about the time that our epidemic began. Viewed according to the seasonal occurrence of our cases, our outbreak may fairly be considered as a sequel to or part of the New York epidemic. In former years our outbreaks have begun in July, never as late as in this year. The coincidence in time with New York State cases just over the border strengthens the belief that our 1916 cases really belong to the New York epidemic.

The outbreak in Vermont in 1916 began a month later than the outbreaks we have had in former years. Our epidemic this year began in August, and reached its height in October.

Furthermore, it should be noted that the first cases occurred during the fourth week of August. The occurrence of the cases from that time until November 1, by weeks, was as follows:—

August 26	6
September 2	5
September 9	4
September 16	3
September 23	9
September 30	4
October 7	7
October 14	6
October 21	4
October 28	5

The following chart shows both the seasonal and geographical distribution of the cases in 1916. (These cases are divided between the east and west side of the natural state division, the Green Mountains.)

SEASONAL AND GEOGRAPHICAL DISTRIBUTION

Counties	<i>East</i>												Total
	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	
Caledonia									2				2
Orange	1								3				4
Orleans		1											1
Washington								1					1
Windham									1				1
Windsor								1		1			2
													<hr/> 11
<i>West</i>													
Addison												3	3
Bennington	1	1						4	3	1			10
Chittenden						1			2	4	1		8
Franklin										1			1
Grand Isle									5	9			14
Lamoille									1				1
Rutland	1							5	3	6	1		16
	<hr/> 3	<hr/> 2	<hr/> 0	<hr/> 0	<hr/> 0	<hr/> 1	<hr/> 0	<hr/> 11	<hr/> 20	<hr/> 22	<hr/> 2	<hr/> 3	<hr/> 53

This year again there is a distinct one-sidedness in the prevalence of infantile paralysis, between the east and west sides of the Green Mountains. As has been before pointed out, these mountains are a distinct barrier to intercourse between the two sides of the state. In epidemics of communicable disease, the effect of this barrier is bound to be observed.

In considering our local outbreak in Vermont for the year 1916, the geographical distribution of the cases emphasizes again the connection of our outbreak with the one in New York City. This year infantile paralysis appeared pretty generally throughout the east. The real epidemic focus was in the Brooklyn Borough of Greater New York. The disease began here early in the summer in June. Dur-

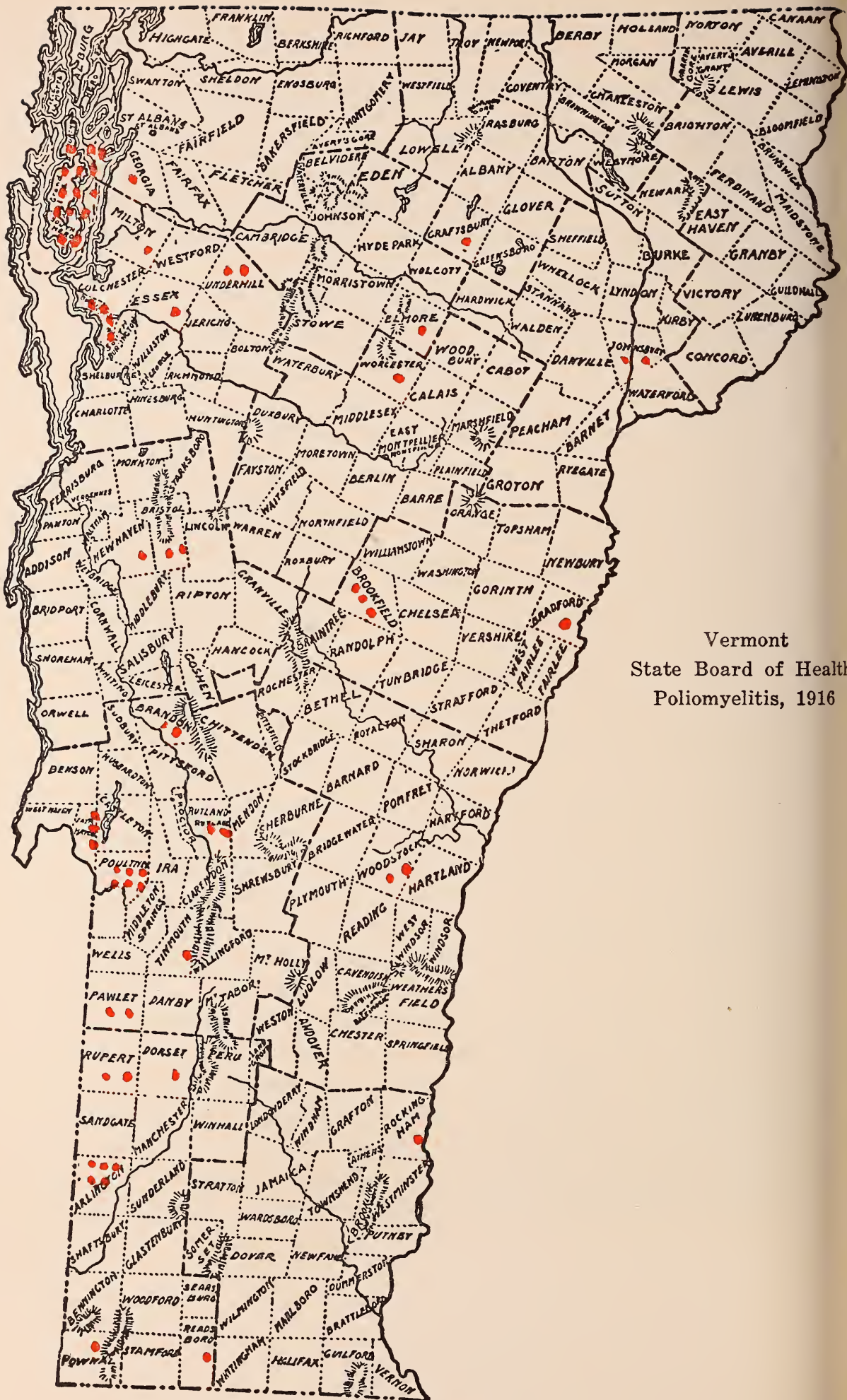
ing July it assumed large proportions and was very evidently reaching out into New York State, Connecticut, northern New Jersey, Massachusetts, and more distant states. The wave did not reach Vermont, however, until late in August.

The epidemic followed generally transportation channels with some notable jumps and exceptions. This is true usually of the disease. Infantile paralysis had been quite general during the winter and spring months. According to the Public Health Reports, 26 states had cases of the disease between January 1 and April 30. The State of Virginia had the most cases. After the New York epidemic started there were many cases, occurring with increasing frequency in 30 states at least. The State of New Jersey had the greatest per capita number of cases of any state in the country for the whole year. The epidemic was especially severe in Jersey City and Newark.

In general, it may be stated that the disease appears to be on the increase throughout the country. It may also be inferred, from the experience of 1916, that the disease is not nearly as much a rural disease as was formerly supposed. During 1916, many large cities suffered severely. In addition to those mentioned, Philadelphia, Toledo, Baltimore, Boston, Chicago, Minneapolis and Providence were seriously invaded. Later in the year, after the disease was on the wane elsewhere, a focus appeared in West Virginia, in which there were 39 cases during the last three months of the year, although it was known that they were not completely reported. Compared with many parts of the east, our Vermont epidemic was mild.

It will be noted by reference to the accompanying map that most of our Vermont cases were clustered in towns close to the New York State border.

Up to August 31, covering the period just before and at the commencement of our outbreak, there had been only



Vermont
State Board of Health
Poliomyelitis, 1916

four cases in Washington County, N. Y., which borders the southeastern counties of Vermont; and none in Essex and Clinton Counties, N. Y., which border the northern counties of our state. Indeed the northeastern section of New York was exceptionally free from cases.

During September, however, there were two cases reported in Essex County, which borders the lower part of Lake Champlain. Fair Haven, Poultney, Pawlet, Rupert and Arlington are all in direct communication by rail with all of eastern New York and there is much interstate automobile traffic between the two states at these points. Burlington and Grand Isle County are in less close contact with New York points by automobile routes and by steam boat. They have, however, close rail connection with the towns in Rutland and Bennington Counties, which were earliest invaded. The earliest cases in our epidemic occurred simultaneously with the cases in the adjoining county in New York and the later and more severe outbreak in Grand Isle County occurred during the same month as the two cases reported in Essex County, N. Y. Clinton and Essex Counties in New York are separated from Burlington and Grand Isle County by the lake and are less closely in touch with the site of our cases in these places than the towns in southwestern Vermont.

MANAGEMENT OF THE OUTBREAK IN VERMONT

From long and varied experience with this disease, we had reason, in Vermont, to anticipate cases, when the very serious outbreak in New York City began to show itself the latter part of June. On July 6, the Board adopted the following regulations relating to children from Greater New York City:

VERMONT STATE BOARD OF HEALTH

QUARANTINE OF CHILDREN FROM NEW YORK CITY

Infantile Paralysis (Epidemic Poliomyelitis) is prevailing to an unusual extent in the city of Greater New York.

Families in that city in which there are children will naturally take such children as far as possible into the country. The State of Vermont has unusual reasons for taking extraordinary precautions against this disease. Infantile Paralysis is generally recognized as a contagious disease and one that may be spread innocently by persons who have no clinical symptoms, in other words, by "abortive cases" or "healthy carriers." The State Board of Health of this State deems it reasonable under the circumstances to make the following rules and regulations; therefore under the authority of Section 5419 of the Vermont statutes, the following rules and regulations are promulgated:

1. No child under the age of fifteen years shall reside in this State for a period of more than twenty-four hours without being reported by an attendant, parent or guardian to the Health Officer of the town or city where such child is, provided such child has been in the city of Greater New York since the 20th of June, 1916.

2. It shall be the duty of every housekeeper, manager or proprietor of every hotel or boarding house where such child is domiciled to immediately report such child, giving the name and age to the Health Officer of his city or town.

3. Every such child shall be subjected to quarantine for a period of two weeks from the time such child was last in the city of Greater New York.

4. The Health Officer of every town and city to whom such a child is reported shall immediately serve a written notice upon the head of the family in which such child is; this written notice shall contain a copy of these regulations and an order signed by such Health Officer requiring such child to remain on the premises in which it is at that time for the specified time of two weeks after last leaving the city of Greater New York.

5. Each Health Officer to whom such child is reported shall require of the attendant, parent or guardian of such

child, a certificate in writing, duly signed by a legal practitioner of medicine, certifying that the nose and throat of such child has been thoroughly washed with a solution of a teaspoonful of common salt in a pint of water, once a day for a period of three consecutive days before the premises are released from quarantine.

6. No child under fifteen years of age shall enter any house so quarantined.

7. A placard containing the word "Quarantine" shall be sufficient evidence to all persons that the premises are quarantined for the purpose of these regulations.

Nothing in these regulations shall be construed to prevent other members of a household in which there is a child as above described, who has left the city of New York since June 20, 1916, from attending to their usual occupations.

The owners, managers or proprietors of hotels and boarding houses may place no restrictions on attendants or guests in their hotels or boarding houses further than the strict isolation of any children as above described from New York City, provided such children are isolated to the satisfaction of the local Health Officer and their noses and throats irrigated as specified.

Each Health Officer will see that a copy of these regulations with which he is furnished is conspicuously displayed in at least three public places in his town or city.

These rules and regulations will remain in force until further notice.

Per Order Vermont State Board of Health.

CHARLES F. DALTON, M.D., Secretary.

Adopted July 6, 1916.

In these regulations, it was sought to restrict the movement of children from the city and as far as possible to prevent their coming in contact, during a possible incubation stage, with other children in this state. Different municipi-

palities in the state adopted these or similar regulations against children from other parts of New York State and other states, as the epidemic spread out to these places from New York City.

On account of the occurrence, during the latter part of August, of cases in the four southern counties of the state, the Board adopted the following regulations with regard to fairs, the annual Rutland Carnival, theatres and picture houses:—

VERMONT STATE BOARD OF HEALTH
REGULATIONS ADOPTED IN REGARD TO THE ATTENDANCE
OF CHILDREN AT PUBLIC GATHERINGS

In accordance with the Vermont Statutes, the following rules and regulations are hereby promulgated by the State Board of Health:

1. All children under fifteen years of age shall be excluded from all fairs. The Rutland Carnival, so-called, shall be abandoned unless effective measures can be taken, satisfactory to the local board of health, by which children under fifteen years of age can be excluded from all public functions, both indoors and out.

2. All motion picture houses and theatres in Rutland, Bennington, Windham and Windsor Counties shall exclude children under fifteen years of age from all entertainments.

3. In towns in which there are one or more cases of infantile paralysis, all children under fifteen years of age may be excluded from all public gatherings, including churches and Sunday schools, in the discretion of the local board of health.

These rules and regulations shall remain in force during the month of September, 1916.

By Order of the State Board of Health.

CHARLES F. DALTON, Secretary.

Adopted August 31, 1916.

These restrictions put upon children from New York City and discouraging the congregation of all children in certain parts of our state apparently accomplished what was intended by the State Board. A large number of New York children came to Vermont during the last days of June and early in July. Yet we had no cases in the state until the last of August. The disease had a good start in our southwestern towns at the end of August, yet there were comparatively few cases, and these scattered, after the promulgation of the last regulations, August 31. The situation at that time was certainly threatening. Reference to the map will show that the cases were confined chiefly to the border towns.

1917

There were 171 cases of infantile paralysis in Vermont this year. There were three scattering cases of the disease in January, an unusual outbreak of six cases in March in Waterbury, and a severe epidemic in Washington County beginning early in June. Beginning with the outbreak in Waterbury in March, Washington County continued to be the center of the 1917 epidemic.

The State Board of Health was well prepared, as in 1916, by reason of its Special Polio Fund, to meet the outbreak of 1917. Dr. Edward Taylor, an experienced and expert diagnostician, devoted himself wholly to the diagnosis, treatment and laboratory research work in the epidemic. Dr. Taylor had had charge of the work in 1916. The unusual clustering of cases in Washington County, especially about Montpelier and Barre, made it possible for him to see a large proportion of all the cases that occurred in the state and to see many of them very early, while they were only suspicious.

As will be noted farther on, many so-called "abortive" cases were diagnosed; and several others, which showed

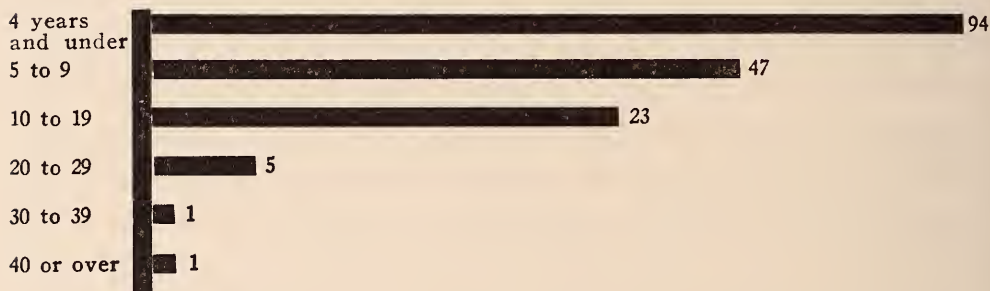
some paralysis later, were also diagnosed in the pre-paralytic stage. In a large proportion of all the cases, lumbar punctures were made and the diagnosis decided by the finding of the microscope cell count and globulin content of the fluid.

INCIDENCE OF THE DISEASE BY AGE AND SEX



Of the 171 cases, 93 were males and 78 females. This division of the sexes does not differ materially from our experience in former years. 54.3 per cent of the cases were males and 45.7 per cent females.

AGE PERIODS 1917



The proportion of young subjects this year was larger than ever occurred in the state before. 54.9 per cent of the cases were four years and under. In 1916, this percentage was 34.3. In the great epidemic of 1914 in Vermont, the proportion of those four and under was only 39 per cent; whereas, in the severe epidemic in New York City in 1916, the percentage of cases under five years was over 80; while in the State of New York, as a whole, the same year, only 49 per cent were under five years of age.

DAY ON WHICH THE PARALYSIS APPEARED

1st day	14 cases
2nd day	28 cases

3rd day	33 cases
4th day	32 cases
5th day	12 cases
6th day	7 cases
7th day	3 cases
After 7th day	10 cases
No paralysis and not stated	32 cases
Total	171 cases

DISTRIBUTION OF THE PARALYSIS

The combinations of muscular paralysis and weakness detected in this outbreak were more varied than ever before. This was due chiefly to the facts that greater pains were taken in seeking out weak muscles and weak groups; and furthermore to the nicer methods of measuring muscular strength or weakness which have been devised by Doctors Lovett and Martin in their clinical orthopedic work in Vermont during the past three years.

Of the 171 cases here recorded, in 139 there was some definite muscular impairment. The following list gives the distribution of the paralysis in these cases, as reported by the attending physicians and health officers. Most of these cases were diagnosed also by Dr. Taylor and many of the reports on muscles involved were afterwards verified by Dr. Lovett.

All the extremities (including abdominal or respiratory)	8
Both arms alone	2
Left arm	4
Right arm	2
"One" arm	1
Left arm and both legs	3
Left arm and left leg	2
Right arm and both legs	1
Right arm and right leg	2
Facial alone	6
Both legs	35
Left leg	20
Right leg	20
Both legs (including abdominal or respiratory)	6
Respiratory alone	4
Both arms (and respiratory)	2
"One" leg	10
Left leg (and abdominal or respiratory)	2
Left arm and right leg	2
Right leg (and abdominal or respiratory)	2

In five instances the location of the paralysis is described as "one leg and facial," "one arm and abdominal," "meningeal type," "both legs and respiratory," "both legs, right arm, and respiratory."

In several instances the exact location of the paralysis is omitted, and 23 cases are reported as "abortive." These latter cases usually occurred in families where there was a frank paralytic case, or, as sometimes happened in Montpelier and Barre, in the immediate neighborhood of such cases. Abdominal muscles were frequently involved in the paralysis. An unusual number of these cases were found at later clinics by Dr. Lovett to have such paralysis, and are not always so classified in our statistics. The cases in this report that are classed as abortive were given both careful clinical examination and examination of the spinal fluid.

There were many more suspicious cases constantly observed in families where there was a paralytic case, which presented very suspicious symptoms. Such cases were frequently diagnosed as "grippy colds," "acute indigestion," "pharyngitis," and there were several cases, attended at first with skin eruptions, which were in several instances diagnosed early as German measles.

RESULTS

The results in these cases, as far as they can be determined from five to eight months after the first symptoms, were as follows:—

Cases with residual paralysis or definite weakness	103
Died	18
Fully recovered	50

171

Most of these cases which still show muscular impairment have been seen at the clinics held by Dr. R. W. Lovett of Boston and the muscular impairment verified by him.

The death rate in this series is remarkably low, viz., 10.5 per cent. The number of males who died was 10, and number of females who died was 8.

The percentage of death in males, therefore, is slightly greater than among the females. The death rate, as a whole, this year is lower than has ever occurred in any outbreak in this state. Our death rate in the epidemic of 1914 was 17 per cent +; the death rate in New York City in the 1916 epidemic was 25 per cent +. The low death rate in this outbreak may be variously explained. There were many slight and abortive cases included in these statistics. There were also a considerable number of cases, which received the most careful attention from Dr. Taylor and some of these were given quite early the immune human serum with apparent gratifying results.

PERCENTAGE OF DEATHS BY AGE PERIODS

Of 94 cases under five years of age . . .	3.19%	died
Of 47 cases from 5 to 9 years of age ..	15.3%	died
Of 23 cases from 10 to 19 years of age..	26.8%	died
Of 5 cases from 20 to 29 years of age	40%	died

These figures are noteworthy simply as showing, in a general way, an exceedingly low death rate. Taken in conjunction with the large percentage of cases that have apparently fully recovered, they emphasize the fact, before stated, that a considerable number of cases were diagnosed in this outbreak, which had comparatively slight paralysis with evident limited nervous lesions, and which, under older and less exact methods, would not have been detected.

The mortality in this outbreak was really even lower than stated, inasmuch as three at least of the 18 fatal cases died so long after the onset of the disease as to make it quite probable that the cause of death was really something other than infantile paralysis. These three cases died from seven to nine weeks after the beginning of the attack. One of these cases is stated to have died of bronchial pneumonia. However, they are classed as fatal cases of infantile paralysis. Omitting these cases from the total deaths, our death-rate would be 8.77 per cent.

CONTACT INFECTION

Infantile paralysis has been usually classed as a disease of low contagiousness. All statistics, even now, tend to confirm this view. This outbreak of 1917 in Vermont was quite carefully studied with reference to possible contagiousness in most of the cases. The following results are recorded:—

Contact with a frank paralytic case, within 2 weeks, 24 cases
 Contact with carrier case, within 2 weeks, 13 cases
 Contact with abortive case, within 2 weeks, 8 cases

A carrier, in this connection, was construed as a healthy person who had been himself in contact with a known case within two weeks. Supposed exposure to an abortive case was an exposure to other persons, usually children, in the same family, who had exhibited within two weeks suspicious symptoms. The more carefully individual cases of this disease are studied, the more are we able to explain their occurrence in this way.

CHILDREN UNDER 20 IN THE SAME FAMILY

1 other child	53 instances
2 other children	40 instances
3 other children	24 instances
4 other children	10 instances
5 other children	7 instances
More than 5 other children	4 instances
"Several" children	2 instances

Here are more than 276 children, in families in which there was one frankly paralytic case; presumably, all these were at some time in contact with this case. Of these children, eight had the disease in paralytic form.

Not all of these eight cases, however, were secondary to the first, as several of these were attacked simultaneously with the first, or within a day or two before or after.

Twenty-two others of these 276 children had abortive attacks of the disease within two weeks, either before or after. Not all of these are included in these statistics. Careful inquiry, however, in most of the families, in which unmistakable cases occurred, brought out the fact that at

some time within two weeks of the case, there had been sickness, frequently of a rather vague character, among other children, when the symptoms presented approximately a picture of the pre-paralytic symptoms in infantile paralysis. These cases were classed as possible abortive cases. For instance, in one family, a child had a sharp febrile attack, attended with vomiting and more or less pain, especially in the head and stiff neck for two days. This was followed in a week by a paralytic case that proved fatal. In another instance, in which there were five other children in the family of a case that was paralyzed, all of the five presented gastro-intestinal symptoms with fever at about the same time.

Of the 276 children included in these statistics, in families where there was a paralytic case, those that were ill, either with frank paralysis or supposed abortive attacks, were not all contact cases with the known infantile paralysis case. They were often taken sick at about the same time with that case. These figures corroborate the statement made before that while the disease is a communicable disease, it is one of low contagiousness.

TONSILS AND ADENOIDS

As far as the statistics of this outbreak were obtained, there were only 16 cases in which tonsils and adenoids were present.

OCCUPATION OF WAGE EARNERS

The occupation of the head of the family in these cases was as follows:—

Blacksmith	4
Clerk	8
Dead	4
Engineer	3
Farmer	27
Fireman	2
Granite cutter	12
Insurance	4
Iron worker	3
Junk dealer	2

Lumberman	4
Lawyer	2
Laborer	8
Machinist	2
Mill work	4
Merchant	3
Painter	2
Printer	2
Paper maker	2
Quarryman	5
"Stone" cutter	21
Traveling salesman	2
Talc miner	2
Veterinary surgeon	2
Not stated	10
Miscellaneous (one each)	31
Total	171

Montpelier and Barre, which were the centers of the disease this year, are granite producing cities. It would be quite natural, of course, that a large proportion of persons in those places affected by any disease should be stone workers. It is, however, a significant fact that of these 171 cases, 38 occurred in the family of persons engaged in the stone industry. In reporting the outbreak of 1913 in this state, which centered about Hardwick, attention was called to the comparatively large number of cases that occurred in that town in the families of granite cutters. No connection has been traced between the stone-cutting industry and the dissemination of this disease. This fact, however, is worthy of repetition; a seemingly large proportion of the cases of infantile paralysis in this state during the last nine years have occurred in the families of those connected with stone working industries.

The population chiefly affected by this outbreak of 1917 in Montpelier and Barre was very cosmopolitan, as shown by the following table:—

NATIONALITY 1917		
	<i>Father</i>	<i>Mother</i>
American	97	95
American-French	2	1
American-Irish	6	9

NATIONALITY 1917—Continued

	<i>Father</i>	<i>Mother</i>
Austrian	0	1
Canadian	3	4
English	3	1
French	11	14
French-Irish	1	0
Irish	3	6
Italian	20	17
Jewish	2	3
Mexican	1	0
Not stated	6	6
Swedish	3	3
Spanish	3	4
Scotch	10	7
	<hr/> 171	<hr/> 171

SEASONAL DISTRIBUTION
1917

The noteworthy fact, brought out by the above chart, is the comparatively early occurrence of the disease this year. The months in which Vermont has chiefly suffered from infantile paralysis have been August, September and October. This year the outbreak may be said to have begun in March, as it is quite probable that there was some connection between the March cases in the town of Waterbury and the later severe outbreak in Barre Town, Waitsfield and Montpelier.

GEOGRAPHICAL DISTRIBUTION

A reference to the map will show that the cases chiefly clustered about the center of the state with Montpelier as a focus. Montpelier, Barre (city and town), Waterbury and Waitsfield were the towns chiefly invaded. St. Albans and Woodstock each had several cases, the latter being the center of a small outbreak. Of all the cases in the state, however, 80 per cent occurred in Washington County; and of the towns of Washington County, the city of Montpelier suffered by far the most severely.

As has been noted, there were six cases in the town of Waterbury in the early spring (March). It was impossible to trace the origin of these cases, occurring at this unusual season, with a monthly temperature mean of 26°.

Waterbury, as far as the records of the Health Department of the state go, had had only three cases of the disease in prior years, viz., 1 in 1912 and 2 in 1914. If the virus may remain dormant under unfavorable conditions for four to six years, as shown by Flexner and Amoss, these prior cases may possibly have given rise to this fresh outbreak. Its importation at this season hardly seems likely. The next cases occurred in Barre Town, Waitsfield and Montpelier. Barre City, it will be remembered, lies between Barre Town and Montpelier. After the Waterbury outbreak in March, no cases were reported in the state until the 7th of June, when a single case occurred in the town of Barre. The next cases occurred the 15th, 16th, 17th, 18th and 19th of June simultaneously in Barre Town, Waitsfield and the City of Montpelier. No explanation of this case in Barre Town is obtainable, further than that the child attended a Decoration Day celebration eight days before.

The cases multiplied very rapidly in Montpelier, Waitsfield and Barre Town simultaneously from the middle of June to the middle of July. Barre City escaped entirely until July 6 when a single case was reported. This case was

one of facial paralysis and was not seen by any physician for three weeks after the development of the paralysis. The only history obtainable in this case was that the child had played with a child in Montpelier the week before the onset of the disease. This case may have infected others, who subsequently developed the disease. Barre City escaped a general epidemic until the middle of the month. From the 16th of July on, scattering cases occurred in the city through August and September. Barre Town, Montpelier City and Waitsfield bore the brunt of the epidemic. These with Waterbury, which was the scene of the March outbreak, were the most severely attacked by the disease. It would be difficult to determine any condition favorable to the spread of infantile paralysis, common to these four towns, that did not also include Northfield, Berlin, Moretown and other neighboring towns. Waitsfield, a small town of 789 inhabitants, twenty miles or so from the railroad, and even further from Montpelier and Barre, had the most cases per capita of population of any town. Berlin, Northfield and Moretown, lying between Waitsfield, Barre and Montpelier, had altogether only two cases, one each in Moretown and Berlin.

The number of cases, per thousand of population, in these towns chiefly affected, was as follows:

Moretown	15.5
Montpelier City	6.8
Barre Town	3.8
Barre City	2.04

The town of Middlesex, lying directly between Waterbury and Montpelier, had but four cases, and these all occurred after August 1, i.e., rather late in the epidemic. The same may be said of the cases that occurred in Woodstock, Pomfret and St. Albans.

The following table shows the number of cases of infan-

tile paralysis that have occurred in Washington County and the whole state during the past eight years.

	<i>State of Vermont</i>	<i>Washington County</i>
1910	69	12
1911	27	1
1912	12	1
1913	47	0
1914	304	25
1915	44	1
1916	64	1
1917	171	137

In all of these years Montpelier City has had but three reported cases of the disease and those in 1910. It will be noted by the above figures that Washington County had a few cases in 1910, more in 1914, and the severe outbreak of 1917, here described, which reached the large proportions of 137 cases; and furthermore, that the City of Montpelier, which was chiefly involved in this year's epidemic, 54 cases, has never before suffered a severe outbreak of infantile paralysis. Basing our prophecy on past experience with this disease, Montpelier and possibly the most of the towns in Washington County will be comparatively immune to infantile paralysis for the next three years.

The map, herewith published, shows that the eastern side of the state, and especially Bennington, Chittenden, Addison and Rutland Counties, almost entirely escaped; also Windham County, Essex, Orleans and Caledonia. In fact, as a general proposition, there was almost none of the disease outside of Washington County. The scattering cases that occurred in Windsor and Franklin Counties might be described, under the old but vague classification, "sporadic" cases.

The following table gives the number of cases in each county by months on the two sides of the state.

1917
East

Counties	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Total
Caledonia									1				1
Essex													0
Orange						1	1		2				4
Orleans									1	1			2
Washington			6			44	32	35	15	5			137
Windham								2					2
Windsor							2	3	8				13
Totals	—	—	6	—	—	45	35	40	27	6	—	—	159

West

Addison	1												1
Bennington													0
Chittenden							1	1					2
Franklin	1							3	2		1		7
Grand Isle													0
Lamoille										2			2
Rutland													0
Totals	2	0	0	0	0	0	1	4	2	2	1	—	12

It is a general observation that infantile paralysis spreads along lines of human communication. Reference has been repeatedly made to the mountain barrier dividing this state. This barrier is very evident from the line of the Winooski River south. Cross state communication north of this river is freer. This explains the comparatively equal distribution of the disease between the east and west sides in 1914—an epidemic general over the northern portion of the state. This has never occurred in the southern half of the state where the mountains form a real barrier to human intercourse. An outbreak in Addison, Rutland and Bennington Counties would not extend to Windham, Windsor, Orange or Washington, or vice versa. Thus with 159 cases this year on the east of the Green Mountains, there were only 12 on the west. In 1910, the east side had 51; the west, 18; in 1911, the west side had 22, the east, 5; in 1913, the east side had 40, the west, 7; and in 1916, the west had 53, and east,



Vermont
State Board of Health
Poliomyelitis, 1917

11. During the past eight years, for which we have records, there have been altogether 23.7 cases per 1000 population on the east side of the state, and 19.5 per 1000 on the west.

The first epidemic in the state, and indeed in this country, occurred on the west side of this state. The western side of Vermont is open chiefly to invasion by infection from New York State and the Province of Quebec. No direct connection with outside epidemics, however, has ever been traced, except in 1916. The eastern side of the state may naturally be invaded from Massachusetts and other New England points, as well as Quebec. Here, too, only one such connection has been traced with certainty, viz: the 1910 outbreak in the Connecticut Valley, which was a direct extension from Springfield, Mass.

In recent years, however, the eastern side of the state has suffered somewhat more than the western, as shown by the above figures.

PREVENTIVE MEASURES

On account of the unusual number of cases being reported from Barre Town and Montpelier (unusual for so early in the summer), the Board met in Montpelier, June 24, and after a conference with the local Board of Health and Mayor and the health officers of some of the neighboring towns advised that all public gatherings in the city be discontinued for the present, and that restrictions be put upon children patronizing ice cream or soda counters. These restrictions were recommended for Montpelier, Barre (Town and City) and Waitsfield.

Cases continuing to appear in these towns, as well as others, especially in Washington County, a meeting of the Board was held in Burlington, July 17, at which Governor Graham was present. At this meeting the following order was made:—

Vermont State Board of Health

Burlington, July 17, 1917.

To date we have had in the state 68 cases of infantile paralysis this year. This is a greater number of cases than we have had any year during the last five years with one exception. Sixty-one of these cases and six deaths have occurred since June 16. These cases with three exceptions have been confined to Washington County. There are good reasons for hoping that this outbreak may be checked at this time. In past years we have had reason to think that large general gatherings of people from many towns have distributed this infection. August and September in past years have been our worst months as far as this disease is concerned.

In view of these facts the attention of local boards of health is hereby called to Act No. 194 of the Laws of 1917 and such boards are directed to make and enforce regulations in the several towns whenever local conditions require such action. When one or more cases develop in any town the local board of health should take action either prohibiting all public gatherings or excluding all children under 16 years of age from such gatherings, also from lunch, soda water and ice cream counters and other public eating and drinking places.

It is hereby ordered that no fairs, Chautauquas, street carnivals or circuses be held in the State of Vermont until further notice.

By order of the State Board of Health,

CHARLES F. DALTON,

Secretary.

As stated in the prelude to this order, it had been strongly suspected in former years, especially in 1914, that the disease was spread by large general gatherings of people, which include children. For this reason, and the further

reason given in the order, that August and September were months in which we had had the most of this disease in former years, it seemed the part of prudence, indeed, imperative, that such general gatherings be dispensed with this year.

There was no serious objection made on the part of any one, as far as known, to the enforcement of this order, except by a company, known as the "Community Chautauqua." Several "Chautauquas" had made contracts in various parts of the state for meetings. The "Community Chautauqua" had apparently the largest number of these contracts. Employing able counsel, this Company secured, through United States District Judge H. B. Howe, an order restraining the State Board of Health and certain local boards from enforcing this order, pending a hearing for a temporary injunction. A hearing was held at St. Johnsbury, July 30, before Judge C. M. Hough of New York City. The decision of Judge Hough is of sufficient importance, from a public health, as well as legal standpoint, so that it is hereby given in full:—

UNITED STATES DISTRICT COURT
DISTRICT OF VERMONT

Community Chautauqua, Inc. vs. Charles S. Caverly, Et Al

Motion to continue restraining order and for preliminary injunction.

Action to restrain the enforcement of an order of the Board of Health of the State of Vermont,—order dated July 17, 1917, and directing that "no fairs, Chautauquas, street carnivals or circuses, be held in the State of Vermont until further notice."

Plaintiff owns and gives that form of entertainment known as a Chautauqua and has made a large number of contracts to give such entertainments in the State of Vermont during the present summer season.

This order of the Board of Health prevents all persons from attending these performances and so in effect prevents the performances themselves. It has been assumed by the parties that the order in question not only does this but terminates the contracts themselves, or so abrogates them as to inflict upon plaintiff a total loss of the amounts expended in preparing to give such entertainments in pursuance of said contracts.

It was admitted that the reason for the order of the Board of Health was, and is, as set forth in the order itself, to wit; a belief that "large general gatherings of people—have distributed" the infection of infantile paralysis.

It was not alleged in the bill, nor urged in argument, that the defendants (who are all the health officers in the State in regions in which it was intended to hold Chautauquas) were actuated by any malice or intended, wantonly or otherwise, to injure plaintiff, but solely by their own opinion of what is desirable from a sanitary and prophylactic standpoint.

Dunnett & Shields for plaintiff.

Herbert G. Barber, Attorney General of Vermont for defendants.

Hough, C. J. The sole ground of jurisdiction set up in this bill is diversity of citizenship and the prayer of the bill is that defendants "be restrained from making or enforcing any order designed to prevent the performance of the contracts" of plaintiff to give Chautauquas in Vermont.

The reason of the bill and this prayer is that the order in question "unjustly discriminates against the plaintiff and against the entertainments to be given by the plaintiff."

It appears from argument, however, that the discrimination complained of is not thought to rest upon any personal prejudice against plaintiff or its entertainments but upon the proposition outlined in the bill, asserted in affidavits, and presented in arguments, that public gatherings in-

duced by an intellectual entertainment such as a Chautauqua do not tend to spread infection and that such is the opinion of some doctors.

Thus the question primarily presented to this court is whether the professional opinion of a board of doctors, honestly exercised, shall be overturned by the chancellor on the ground that it is "unreasonable."

It is said that *State vs. Speyer*, 67 Vt. 502, upholds this view.

Undoubtedly there are cases (and the case cited is one of them) wherein police regulations cannot be held justifiable "unless there are reasonable grounds for a belief that the necessary protection of the public health" requires their passage. This is a simple doctrine and means no more than that it is the duty of the court to examine into the facts of every case and if a responsible, honest and presumably reasonable body of professional opinion is found on the side of the regulation, it is the duty of the Court to uphold it even though the chancellor should entertain the view of professional dissidents.

The point is not whether the court agrees with the professional conclusion of a body of doctors, or engineers, or clergymen, but whether it is evident that the professional view is a reasonable view for men of the proper profession to entertain. They may be wrong, but is there any reasonable probability of their being right? If that question is answered in the affirmative, the professional regulation cannot be said to be unreasonable,—as a matter of law. This is the view taken in the *State vs. Morse*, 84 Vt. 387, and the whole matter is covered by the remarks of Holmes, J., quoted (at page 397) from *Laurel Hill Cemetery vs. San Francisco*, 216 U. S. 358.

I am, therefore, not called upon to come to any conclusion as to whether the propagation of poliomyelitis is actually assisted by crowds, but I am persuaded; (1) that a very

responsible body of professional opinion, is that way; (2) that the Vermont Board of Health shares that view; (3) that it has just as much right to entertain that view as I have to entertain an opinion upon a point of law; and (4) that such a point of view cannot be held to be unreasonable.

Although the bill does not in terms rest upon any constitutional point, such point was necessarily presented.

Thus the bill prays to have certain contracts preserved,—preserved from what? From an exercise of the police power of the State in accordance with a responsible body of professional opinion.

An act of the Legislature thus impairing a contract is not unconstitutional. *Manigault vs. Ward*, 123 F. R., 707; affirmed 199 U. S. 473.

That the prevention of disease, or its spread, by any means based on responsible medical opinion is a competent and constitutional exercise of police power is a proposition so plain as to scarcely require citation.

The Legislature might have said that there should be no gatherings at all except by license (*Davis vs. Commonwealth*, 167 U. S. 43) and discrimination or classification is frequently based not on medical opinion but merely on matters of taste. Of this, perhaps the best illustrations are the "Ice cream cases," of which *Powell vs. Pennsylvania*, 127 U. S. 678 was the first (and the last is as yet unreported).

That the Board of Health acted within its statutory authority is, I think, plain from Chapter 194 Vermont Laws of 1917 which explicitly authorizes local health officers (such as most of the defendants herein) to "forbid and prevent the assembling of people in any place, when the State Board of Health deems that the public Health and safety so demand."

This is not a delegation of law making authority, for the Assembly laid down the law but entrusted its application

to medical men who would be presumed better informed as to local conditions.

If this matter be regarded as one of local law, only cognizable in the United States Courts because of diversity of citizenship, I think the matter fairly within the ruling in *state vs. Morse supra*; if (looking beyond the form of pleading) other questions be considered, no constitutional rights of plaintiff have been invaded. Therefore, the application cannot be granted,—a result the more willingly reached because the papers presented (especially the results of poliomyelitis observations in Vermont for some years past) conclusively show to me that the moves of this mysterious disease are so little understood that any honest medical efforts to effect its extermination should meet with assistance rather than hostility.

The restraining order is dissolved and preliminary injunction denied.

A motion to amend the bill was made at the hearing and no objection made thereto. If the form of the amendment is transmitted to me through the clerk of the Court, it will be formally allowed so far as can now be seen.

July 30, 1917.

C. M. HOUGH,

(Endorsed) Filed August 3, 1917.

Cir. Judge.

FREDERICK S. PLATT, Clerk.

UNITED STATES OF AMERICA, DISTRICT OF VERMONT

I, Frederick S. Platt, Clerk of the District Court of the United States, within and for the District of Vermont, hereby certify that the foregoing is a true and complete copy of the original opinion and order made, filed and docketed in cause No. 47 on the Equity Docket of said Court, entitled:

Community Chautauquas, Inc.

vs.

Charles S. Caverly, Et Al.

Witness My Hand, as such clerk and the seal of said Court, at the office of the Clerk of said Court, in the City of Rutland, in said District this 18th day of March, A. D., 1918.

(Signed) FREDERICK S. PLATT,

Clerk.

On the blanks used in collecting data of this outbreak, there is this question: Has patient been to any large public gathering? If so, place and date.

While this question is often unanswered, the answers given show that fourteen children attended such public gatherings within two weeks before being taken sick. Nine of these were outdoor functions, such as Decoration Day or Fourth of July celebrations or school picnics, and five had attended picture shows.

Meantime many towns and cities in the state had taken advantage of an Act of the last Legislature, which authorized Health Officers to "order churches, schools, and all places of public entertainment to be closed"; and to "forbid and prevent the assembling of people in any place, when the State Board of Health deems that the public health and safety so demand."

The same Act provided that "the local Board of Health in a town or city may make and enforce Rules and Regulations in such town or city, relating to the protection of the public against contagious and infectious diseases and the cause, development and spread of any disease, provided such Rules and Regulations have the approval of the State Board of Health."

Some of these towns were Barre City, Braintree, Shelburne, Woodbury, Northfield, Richmond, Randolph, Rutland City, Duxbury, East Montpelier, Greensboro, Bradford, Brookfield, and Weathersfield. The regulations adopted by these towns were quite similar. A sample is herewith given:

Braintree, Vermont, July 17, 1917.

In accordance with No. 194 of the laws of 1917, the local board of health of the town of Braintree hereby makes the following rules and regulations against the cause, development and spread of infantile paralysis.

1. Children under 16 years of age entering this town from any town or city where the disease is known to exist shall be subject to quarantine for the period of two weeks.

2. Children under 16 years of age shall be excluded from all public gatherings held indoors or outdoors, including churches, Sunday schools, theatres, places of amusement and all public gatherings of any kind.

3. Children under 16 years of age shall not be served at any ice cream counter, soda water fountain, restaurant or any place where food or drink is sold to be consumed on the premises, exception being made to hotels.

These regulations shall continue in force until vacated by a written order signed by the Secretary of the State Board of Health or the local health officer.

Local Health Officer.

Approved by the State Board of Health.

Secretary

To

.....

You are hereby notified of the above rules and regulations and ordered to observe the same.

Signed

Health Officer for June, 1917

While, as has been stated, the disease, infantile paralysis, is undoubtedly of low contagiousness, and not at all comparable in this respect with such diseases as measles or smallpox, there is no doubt that the main, if not the only, method of distributing the disease is through human agencies. *Human beings*, rather than *things*, *animals*, or *insects* are without any doubt the chief distributors of the disease.

Furthermore, it is well established that not only frankly paralytic cases, and the so-called abortive cases, but also healthy persons may harbor and distribute the virulent organisms of the disease.

If these facts *are* facts, they place a distinct duty upon health officials; that duty involves the prevention of general gatherings of human beings in the presence of this disease in epidemic form. The public gatherings directly affected by the original order of the State Board were such as would be likely to attract people from widely separated districts. Washington County, the center of the disease this year, is in the center of the state. There is probably not a county in the state, in which, if large meetings of any kind were held, there would not be representatives from this county.

At the middle of July, 1917, there were over 50 cases of this disease, and confined almost exclusively to this county. The restrictive measures adopted by the State Board and by local boards in various towns were apparently justified. Reference to the map, showing the distribution of the disease through the year, seems to prove this. The disease in epidemic form was confined to Washington County. Scattering, "sporadic," cases occurred in several counties; but these are not only such as are quite likely to occur in any year, but there was nowhere else anything approaching epidemic conditions.

Among all the towns, excepting Barre City, that took advantage of the statute authorizing their local boards to make and enforce Rules and Regulations against contagious and infectious diseases in this outbreak, there were only three cases of the disease after the promulgation of such Regulations.

The experience of Barre City in this epidemic is interesting. As has been stated, this city, which is much the largest place in Washington County, was surrounded by the disease in June; Barre Town and Montpelier having each

many cases from the middle of that month. A single case, which was probably of at least two weeks' standing, was discovered in Barre City, July 6. There were no more cases in that city for ten days. It cannot be stated with any certainty whether the first of the cases in the real epidemic in that city were any of them traceable to this case that went undiscovered until July 6.

Barre City had early taken precautions recommended by the State Board of Health on June 24, at the meeting held in Montpelier. These regulations had been quite carefully enforced and it was a matter of comment at the time that Barre City was escaping the general outbreak affecting surrounding towns. This was especially noticeable because of the intimate relations existing, socially and commercially, between Montpelier and Barre. Closely related in this way and with free communication by way of trolley, steam cars, and highway, it was naturally expected that Barre City would have cases of the disease early. When finally it became evident that this city was sharing in the epidemic of the surrounding towns, the local Board of Health adopted the following stringent Regulations:

Barre, Vermont.

In accordance with No. 194 of the laws of 1917, the local board of health of Barre, Vt., hereby makes the following regulations against the cause, development and spread of infantile paralysis.

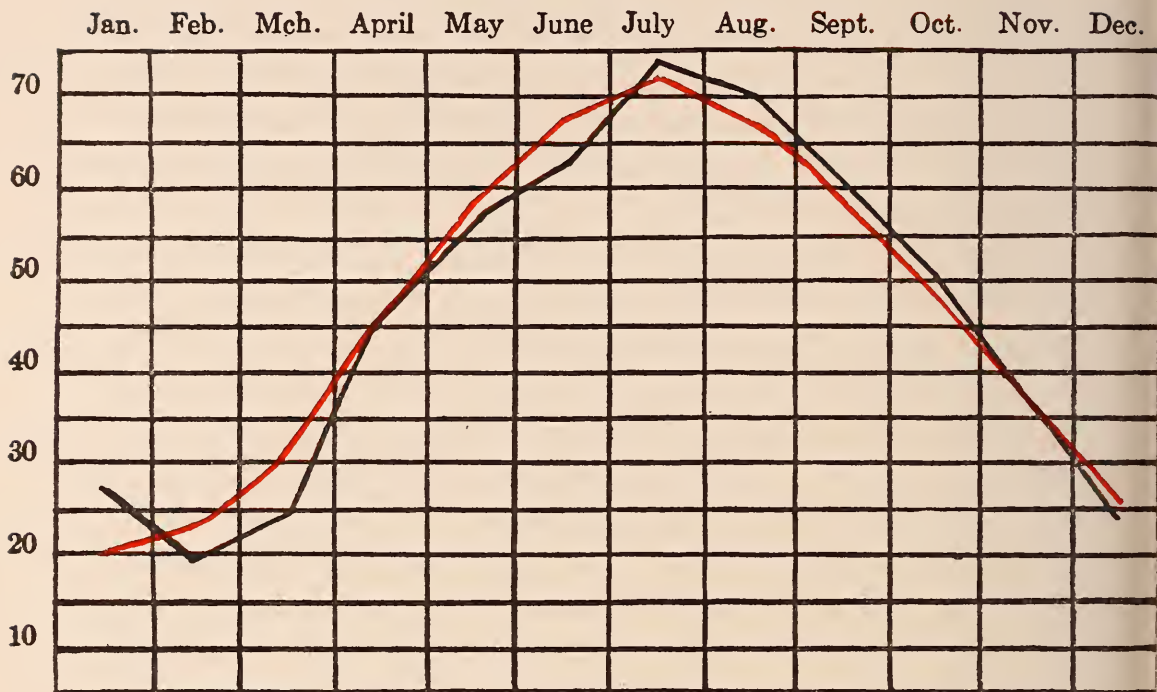
1. All public gatherings shall be prohibited, whether they are to be held indoors or outdoors, including schools, Sunday schools, churches, theatres, picnics, ball games, first class saloons, lodge meetings, club rooms, pool rooms, soda fountains and ice cream parlors, if ice cream is to be consumed on premises.

2. All children under 16 years of age shall be kept on their own premises, except those who are working.

TEMPERATURE CHART. Northfield, Vt., 1916

Normal—35 Years —————

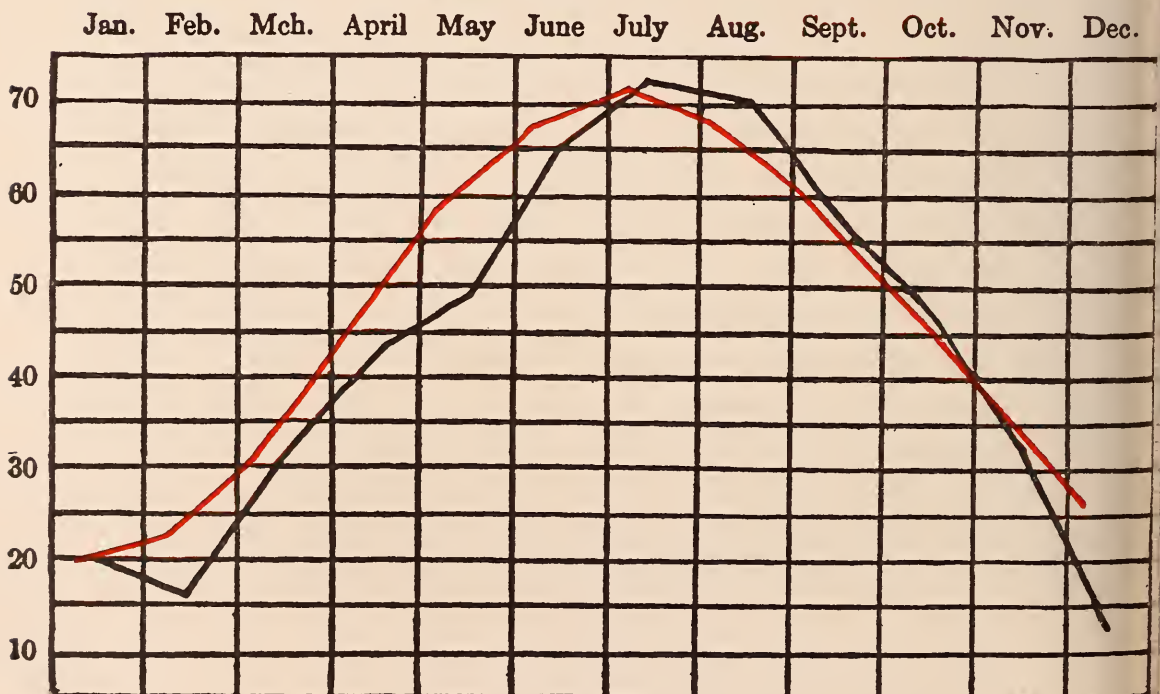
Mean—1916 —————



TEMPERATURE CHART. Northfield, Vt., 1917

Normal—36 Years —————

Mean—1917 —————



To be in force until vacated by written order signed by Secretary of Board of Health.

Thereafter only 16 cases were reported in that city. Some objection was raised to the somewhat drastic Regulations referred to. An injunction was secured from a Judge of the Superior Court, restraining the local Board of Health from interfering with the conduct of its business, by one firm. A hearing was promptly held and the injunction dissolved. Thereafter, the Regulations of the local board were strictly and generally enforced. Barre City's comparative exemption from the disease is undoubtedly largely due to the stringent measures adopted by the local board.

WEATHER: 1916 AND 1917

Heretofore, the waves of infantile paralysis in Vermont have generally coincided with *dry* weather. *High temperatures* have not been noticeable.

The United States Weather Bureau, located at Northfield, is in the immediate neighborhood of the epidemic of 1917. It will be observed by the following charts that the July and August temperatures in each year were somewhat above the normal, but that the temperatures for the early summer months, May and June, and in the case of 1917, April also, are below the normal.

The mean temperature for the year 1916 shows a 0.3 departure; and, in 1917, a 3.2 departure. These two years, therefore, especially 1917, could not be considered hot years. March had a mean temperature of 26°. A recent winter outbreak in West Virginia in December and January occurred with a mean temperature for the period of 32. (Draper.)

The chart on page 189, which is a continuation of the one published in the report for 1914 and '15, shows graphically the precipitation departures for the series of twelve years. This chart was prepared to study the relation which our infantile paralysis outbreaks bore to *dry* seasons. It in-

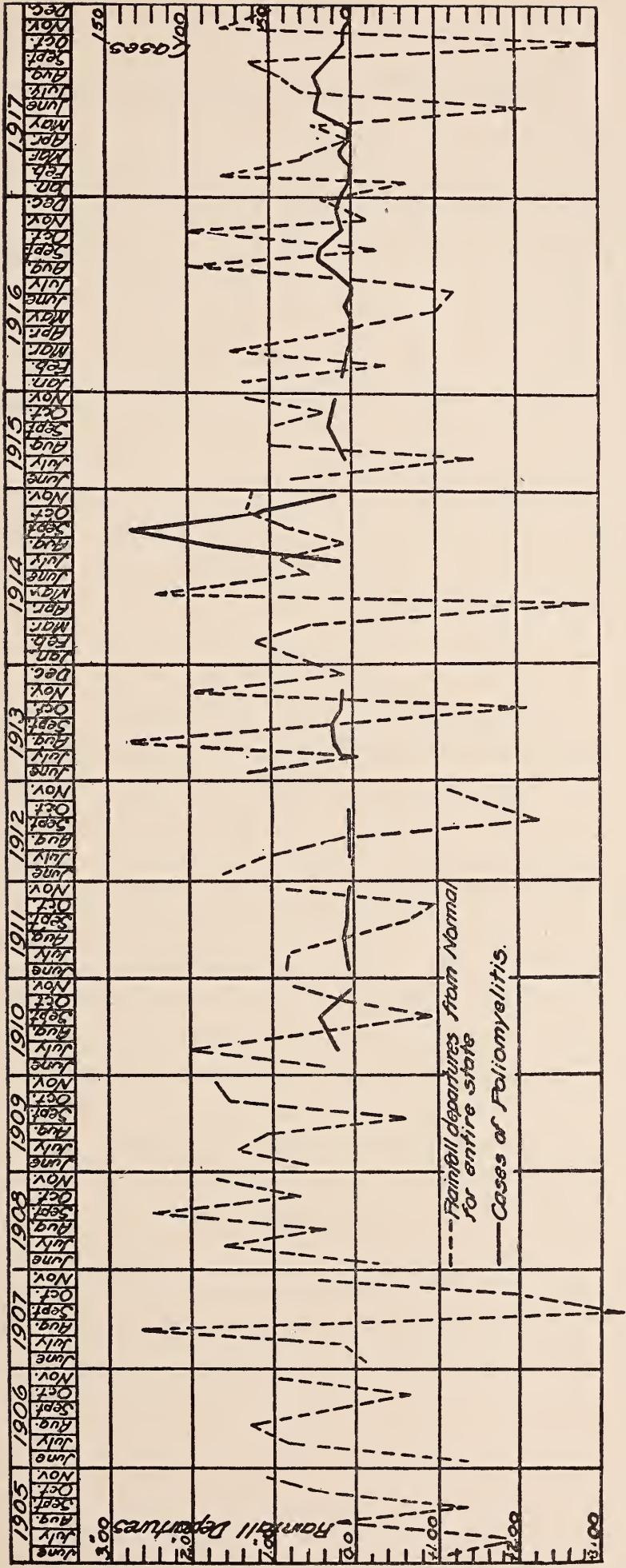
cludes only the infantile paralysis months of the first eleven years. The dotted lines show the rain fall departures; the part above zero line representing dryness, and the part below the zero line representing the excess of rainfall. Both 1916 and 1917 were dry years. In 1916, this was especially true of March, August and October; whereas, May, June and July were wet months. The figures for 1917 show a minus departure, though less than 1916. June and October were wet, whereas July, August, September and November were dry. On the whole, it may be said from the data obtainable from the Weather Bureau that "seasonal dryness" in these two years was a fact. While this is a fact, it must be said that the coincidence of the severity of our outbreaks of this disease and *dry* seasons is not striking.

POLIOMYELITIS WORK DONE BY THE BOARD UNDER THE SPECIAL FUND

The philanthropic work, anonymously supported, to which reference was made in the last Biennial Report of this Board, has been continued during 1916 and 1917. The Research Laboratory has been maintained and the after-treatment of the cases has gone on—both with highly gratifying results.

Laboratory

Carrying out the original plan of the anonymous donor, a portion of the fund was set aside for laboratory purposes. In order that the entire attention of the laboratory should be concentrated on poliomyelitis, a separate organization was made and the laboratory established with the cooperation of the University. The laboratory occupies four rooms on the ground floor of the College of Medicine and has an animal room on the roof. The equipment includes special apparatus for diagnostic work and for investigation of filterable viruses.



Personnel

The Advisory Board consists of Dr. Charles S. Caverly, President of the State Board of Health; Dr. Simon Flexner, Director of the Rockefeller Institute for Medical Research and Dr. B. H. Stone, Director of the Laboratory of Hygiene of the State Board of Health.

Dr. Harold L. Amoss of the Rockefeller Institute for Medical Research has directed the activities of the laboratory. Dr. Edward Taylor of Alabama has continued director of the Research Laboratory.

The laboratory has three functions: first, differential diagnosis of cases of poliomyelitis; second, the specific treatment of cases; third, investigations to add to our knowledge of the disease.

Diagnostic Work

On early diagnosis depends not only success in treatment but the greater problem of prevention. Amoss and Chesney have shown that cases of poliomyelitis treated with human convalescent serum before the forty-eighth hour of illness have greater chances of recovery. Persons coming into contact with active cases of the disease may be regarded as potential carriers, for it has been shown that persons apparently well may harbor the virus or active infecting agent in their nasal secretions. It becomes imperative, therefore, to establish early diagnosis. Early diagnosis, even before onset of paralysis, can be made, but since there is no definite biological reaction by which a diagnosis can be made it may be an exceedingly difficult task. The chemical bacteriological and cytological examinations of the spinal fluid give invaluable assistance in diagnosis but the results must be interpreted in the light of clinical examination. Accurate differential diagnosis is best made by the physician who has both laboratory and clinical training at his command. Since the average physician is not constantly called upon to diag-

nose poliomyelitis, it becomes advisable to centralize the responsibility of diagnosis in such a laboratory. During the summer season, most of the time of the laboratory staff was occupied in diagnoses. During 1915, 1916 and 1917, many cases were visited and 281 diagnosed as poliomyelitis.

In addition to the value of diagnostic activities already referred to, the educational value of such a method should be borne in mind. Clinics have been held in various parts of the state and to more than 125 physicians have been demonstrated lumbar puncture and method of examination of the fluid, together with the methods of clinical examination. This part of the work of the laboratory has undoubtedly resulted in a decrease in the number of cases from the expectant rate. During 1914, there were 306 cases; in 1915, 44 cases; in 1916, 64 cases, the time of the great New York epidemic. As one result of the laboratory, several localized epidemics were apparently stopped with the appearance of the first series of contact cases. Due to accurate quarantine, no second contact cases occurred.

Treatment

With an additional fund of \$1,000 for expenses contributed by Dr. F. S. Lee of New York, it was possible in 1916 to pay convalescent patients for serum to be used in the treatment of acute cases. It became the duty of the laboratory to collect the serum and to administer it at the homes of new patients throughout the state. During 1916, 19 patients were thus treated. Of these, two died and in two the infection was not stayed and in one case no improvement observed. Marked improvement, however, was obtained in fourteen of the cases treated. These cases have been reported in the New York Medical Journal for May, 1917, by the Director of the Laboratory, Dr. Taylor.

During 1917, 11 cases were treated, with two deaths. In the two years 1169 c.c. of serum were administered. (1916, 779 c.c.; 1917, 390 c.c.)

The cases were treated with large amounts of serum intraspinally and intravenously, according to the method of Amoss and Chesney. It was found that the serum remained potent after preservation with 0.25 per cent trikesal at the temperature of the ice-box for at least nine months. The serum should, however, be used as early as possible after withdrawal from the patient.

The results obtained confirm those of previous workers and demonstrate the practicability of carrying on such treatment in the field.

Research

With the yearly recurrence of cases within the state, ideal opportunity offered itself for investigation. Many workers, some with large endowments, have been engaged in research in poliomyelitis over a number of years. Moreover, the only experimental animal thus far found to be susceptible to infection with poliomyelitis is the monkey, making experimental work very expensive and slow. In spite of these apparent difficulties the results of two years' work have been unexpectedly gratifying. Two reports published in the *Journal of Experimental Medicine* have contained noteworthy contributions to our knowledge of the disease.

Naturally, the investigator's endeavors were directed along lines which promised practical results. Among the first studies was the improvement of methods for the detection of carriers of poliomyelitis. Epidemiological observations first by Wickman pointed certainly to the existence of human carriers who showed no clinical evidence of further invasion of the virus. Swedish and American observers have proven experimentally by the inoculation of filtered nasal secretions from contacts that healthy persons sometimes carry the virus, but out of many attempts only a small percentage of the positive results were obtained. This may be explained by the natural difficulties attending the

isolation of the virus from such sources. Monkey inoculation is the only method at our command for detecting the virus, and virus of human origin possesses relatively low infective power for monkeys. Moreover, the virus is usually present in the nasal secretions in small amounts and thorough rinsing of the nasal cavities results in relatively large amount of fluid. This fluid is contaminated by many organisms, usually found in the nose, and which must be removed by filtration before injecting into the monkey. Route of infection may be induced in a monkey by the smallest amount of virus when the direct intracerebral route is used. From four to six c.c. of fluid can be safely injected into the monkey brain but the nasal washings from a single person may be fifteen to twenty times as much. Efforts to overcome some of these resulted in a method, roughly, five times as sensitive as the former methods used. The improved method takes advantage of the solubility of mucin in sodium bicarbonate solution. After adding sodium bicarbonate and shaking with glass beads, the fluid is filtered and rapidly reduced in volume at 35 deg. C. under reduced pressure. The residue is dialyzed and injected intracerebrally into the monkey. One of the particular points is the rapid handling of the nasal secretions after collection.

In testing the method on controls, it soon becomes evident that still another factor operated to decrease the number of positive results if washings from more than one person were mixed. Sterilized nasal secretions from various persons do not react alike when incubated with the virus. The results of 62 experiments show that nasal washings possess definite power to neutralize the active virus of poliomyelitis. This contribution of our Research Laboratory is one of the most important additions yet made to our knowledge of infantile paralysis. This power of naso-pharyngeal muc-

cus to neutralize poliomyelitis virus is not absolutely fixed but is subject to fluctuation in a given person.

Apparently, inflammatory conditions of the upper air passages tend to remove or diminish the power of neutralization, but irregularities have been noted even in the absence of these conditions. These experiments suggest that the nasal washings of *children* possess the neutralizing power less than those of *adults* and also that the neutralizing power is diminished in the *summer months*. The neutralizing substance is water-soluble and appears to be due to salts; it appears to be slightly changed by heat and does not depend upon the action of mucin, as such. Experiments are under way to determine definitely whether seasonal variations occur and to determine the distribution of the neutralizing power among definite groups of persons. This neutralizing action of the nasal secretions is suggested as one of the factors determining whether or not persons exposed become carriers of the disease, if they have other mechanisms of defense or contract the disease in the absence of other means of defense.

The second contribution from the laboratory deals with the mode of infection of poliomyelitis. The clinical observations of the Swedish observer Wickman strongly suggest that the microbic cause of poliomyelitis may be transmitted by apparently healthy carriers. The production of experimental poliomyelitis in the monkey by the injection of nasal secretions from persons who had been in contact with cases of the disease definitely established the carriage of the virus. But the observations by Flexner and Lewis, and Kling and Peterson have been too few to convince many observers of the validity of the theory. During 1917, the Research Laboratory studied the prevalence of carriers in Vermont. The report of examinations of a family of four children published in the *Journal of Experimental Medicine* forms an instructive illustration of the mode of infection of the

disease as brought out by clinical and experimental study. This study establishes more firmly the carriage method and describes for the first time the occurrence of two carriers in the same family and carriage of the virus during the incubation period of the disease. A family group containing four children, all of whom showed in varying degree symptoms of poliomyelitis, is described. The source of infection and periods of incubation have been followed. Two of the children were proven by inoculation tests to carry the virus of poliomyelitis in the nasopharynx. Of these, one was detected to be a carrier after recovering from a non-paralytic attack of the disease, and the other was discovered to be a carrier about five days before the initial symptoms, attended later by paralysis, appeared. The original case from which the three others took origin was fatal; the youngest child, after quite a severe onset, was treated with immune serum, and made a prompt and almost perfect recovery. The nasopharyngeal secretions of two of the cases, taken one month after the attack, proved incapable of neutralizing an active poliomyelitic virus.

The proposition is presented that every case of poliomyelitis develops from a carrier of the microbic cause, or virus, of poliomyelitis.

The laboratory is now studying the question of selective communicability.

TREATMENT OF RESULTANT PARALYSIS

"The Vermont Plan"

Dr. Robert W. Lovett, Orthopedic Surgeon to the Children's Hospital, Boston, and Professor of Orthopedic Surgery at Harvard Medical School, still directs this work. Dr. Lovett was assisted at his earlier clinics by his assistant in private work, Miss Wilhelmine G. Wright. He began his clinics in December, 1914. These clinics were repeated the following summer (1915); also in 1916 and 1917 in the

summer. They were held at points in the state which seemed best adapted to accommodating the patients, viz., Burlington, Rutland, Montpelier, Barton and St. Albans. These clinics were held at the local hospitals, except in the case of Barton, which has none. The medical profession generally and nurses were always invited and many always were present. Besides Miss Wright, Dr. Lovett had the assistance of Miss Janet B. Merrill, Miss Helen King and Miss Rebecca Selfridge, all trained under his direction for this special work, at his later clinics. These women were all employed later for supervision work in the field.

Reference was made in the last report to a method of measuring muscular strength by means of the spring balance, which was devised for and first tested on these Vermont cases. This method, devised by Dr. Lovett, in conjunction with Dr. E. G. Martin of the Physiological Department at Harvard has proven of much practical value.

This plan of holding free public clinics for the maimed children, following an outbreak of Infantile Paralysis, has been since adopted in other states, notably New York, after the 1916 epidemic, and has become known as "The Vermont Plan."

CENSUS OF CASES SEEN AT VARIOUS CLINICS

Number of cases seen at the Dec., 1914 and Jan., 1915 clinics, 212:

Burlington	47
Montpelier	41
Barton	45
Rutland	41
St. Albans	38
<hr/>	
Total cases	212

Number of cases seen at the July, 1915 clinics	122
Burlington (18 new,—19 Jan., 1915)	37
Montpelier (7 new,—10 Jan., 1915)	17
Barton (16 new,—12 Jan., 1915)	18
Rutland (12 new,—11 Jan., 1915)	23
St. Albans (7 new,—20 Jan., 1915)	27
<hr/>	
Total cases	122

Number of cases seen at the July, 1916 clinics	108
Burlington	34
(12 new, 4 Jan., 1915, 2 July, 1915, 16 Jan. and July, 1915.)	
Montpelier	10
(2 new, 5 Jan., 1915, 1 July, 1915, 2 Jan. and July, 1915.)	
Barton	21
(12 new, 4 Jan., 1915, Jan. and July, 1915.)	
Rutland	23
(7 new, 5 Jan., 1915, 4 July, 1915, 7 Jan. and July, 1915.)	
St. Albans	20
(3 new, 3 Jan., 1915, 3 July, 1915, 11 Jan. and July, 1915.)	
<hr/>	
Total cases	108

Number of cases seen at the August, 1917 clinics	186
Burlington	45
(21 new, 5 Jan., 1915, 4 July, 1915, 3 July, 1916, 2 Jan., 1915 and July, 1916, and 10 all clinics.)	

Montpelier	56
(44 new, 2 Jan., 1915, 2 July, 1915, 1 July, 1916, 6 Jan. and July, 1915, 1 Jan. 1915 and July, 1916.)	
Barton	25
(7 new, 4 Jan., 1915, 1 July, 1916, 3 Jan. and July, 1915, 3 Jan., 1915 and July, 1916, and 7 all clinics.)	
Rutland	38
(16 new, 2 Jan., 1915, 2 July, 1915, 2 July, 1916, 2 Jan. and July, 1915, 3 Jan. 1915 and July, 1916, and 8 all clinics.)	
St. Albans	22
(2 new, 2 Jan., 1915, 2 July, 1916, 4 Jan. and July, 1915, 2 Jan., 1915 and July, 1916, 1 July 1915 and July, 1916, and 9 all clinics.)	
<hr/>	
Total cases	186
Total number of cases seen at all the clinics	628
Individual cases	392

SUMMARY OF RESULTS OBTAINED—AMONG THE INDIVIDUAL
CASES SEEN IN 1914, 1915 AND 1916

(Embracing cases supervised by Miss Merrill and Miss King, outside of Washington County.)

Followed treatment	96
Improved	74
No change	3
Worse	0
Complete recovery	5
Practical recovery	14
<hr/>	
	96
Followed treatment irregularly	37
Improved	18
No change	16
Worse	3
Complete recovery	0
Practical recovery	0
<hr/>	
	37

Number who did not follow treatment	50
Improved	6
No change	20
Worse	23
Complete recovery	0
Practical recovery	1
	<hr/>
Total	183

1917 CASES, EXCLUSIVE OF THOSE SUPERVISED BY MISS SELFRIDGE
IN WASHINGTON COUNTY

Followed treatment	102
Improved	82
No change	5
Worse	0
Complete recovery	4
Practical recovery	11
	<hr/>
	102
Followed treatment irregularly	29
Improved	16
No change	12
Worse	1
Complete recovery	0
Practical recovery	0
	<hr/>
	29
Number who did not follow treatment	37
Improved	6
No change	22
Worse	8
Complete recovery	0
Practical recovery	1
	<hr/>
	37
Total	168
	183
	<hr/>
	351

To obviate a common error, in practicing massage, using electricity, allowing walking or other natural exercises too early, Dr. Lovett, after his examination of a large number of recent cases in Washington County in 1917, suggested that many of these be placed under early and continuous supervision. Miss Selfridge was given charge of this work, and saw these cases in Barre City, Montpelier and Waitsfield twice a week, supervising the training of the weak muscles.

Her report (to May, 1918) follows:—

Number followed treatment regularly	41
a. Complete recovery	12
b. Practical recovery	9
c. Improved	18
d. No change	2
e. Worse	0
	<hr/>
	41
Number followed treatment irregularly	31
a. Complete recovery	1
b. Practical recovery	4
c. Improved	23
d. No change	3
e. Worse	0
	<hr/>
	31

The gratifying results as indicated by these figures suggest possibilities in the future management of these cases, of greatly minimizing the permanent paralysis. These results show the importance of early and careful supervision. This supervision simply ensures *rest* of the child and especially the affected limbs or muscles until muscle-training can be safely begun. It then directs this training.

Besides the advice given these cases at the clinics and the field work done by these nurses, many cases have been placed in Vermont hospitals, or the Children's Hospital in Boston for surgical treatment, the expense being met out of our "Special Fund."

During the last two years, 114 pieces of apparatus, braces, corsets, splints, etc., have been provided for children in the state. Since the beginning of this work in 1914, 168 such pieces of apparatus have been secured and applied to these cases. The expense for this has been met in part by the patients and their friends and, where necessary, has been paid from our Special Fund.

THE TREATMENT OF INFANTILE PARALYSIS

PRELIMINARY REPORT BASED ON A STUDY OF THE VERMONT EPIDEMIC OF 1914*

R. W. LOVETT, M.D., Boston

PRIOR to the year 1907, infantile paralysis was rather an uncommon affection in this country. The severe New York epidemic of that year was followed in the two succeeding years by a great increase in the number of cases throughout the country, and since 1909 the disease has each summer claimed thousands of victims. As a result of this condition, clinical opportunities have come so fast that therapeutic knowledge has not been able to keep pace with them. Scientific knowledge of the disease in these years has also made great strides. The nature of the affection and its organism have been identified; its pathology has been cleared up experimentally in animals and by necropsy in the human being; and knowledge of symptomatology and prognosis has been greatly enlarged. But in the matter of therapeutics, in early cases especially, although we have made progress, we have made no great strides forward, and although cases on the whole are treated much better than they were ten years ago, we have today a very crude knowledge of the real value of the therapeutic measures at our disposal, and of their proper application and limitations. Electricity and massage are much used, but not always intelligently. The proper dosage is uncertain, and rests on an empiric basis. Muscle training is of undoubted value. We all believe in the use of affected muscles

*Copyright, 1915, Am. Med. Ass'n, and reprinted by permission from *The Journal of the American Medical Association*, June 26, 1915, Vol. LXIV, pp. 2118-2123.

so far as possible, but we know little of the effect of muscular fatigue on the paralyzed or partly paralyzed members. Much more exact knowledge in the lines indicated must be acquired before the best functional results can be obtained from treatment.

It has been generally assumed that the distribution and the extent of the ultimate paralysis are wholly determined by the location of the lesions in the cord, but certain clinical aspects of the Vermont cases suggest that possibly there is another factor in this determination—that of muscle function. The records of these cases have therefore been analyzed, as a result of which the ground is taken in this paper that although infantile paralysis is apparently a widely distributed and indiscriminate lesion of the cells of the anterior cornu of the spinal cord, yet it would appear that the susceptibility of some motor centers was greater than that of others, influencing perhaps localization, and secondly, that the amount of residual paralysis was not wholly determined by the cord localization, but also influenced to some extent by the function of the affected muscles.

The following preliminary report of the observation of a group of cases is offered in the hope that it may throw some light on certain phenomena of the affection, and certain deductions as to treatment are added, made from the observation of these cases and of others. Certain investigations now being made at the Children's Hospital by the physiologic department of Harvard University will, it is hoped, throw light on the value of some of our therapeutic measures.

In the fall of 1914, I was asked by the State Board of Health of Vermont if I would undertake on their behalf the treatment of the cases of infantile paralysis occurring there in the summer of 1914, of which there had been 306. A private citizen had given to the State Board of Health a certain sum of money to be expended on an investigation into the

epidemiology of the epidemic and on the treatment of the affected persons. Dr. Simon Flexner of the Rockefeller Institute consented to take charge of the epidemiology end of the inquiry, and I embarked on the enterprise of the treatment of these cases in December, 1914.

The conclusions as to the epidemiology, occurrence, contagiousness, etc., I have left entirely to the other side of the investigation, and have confined myself strictly to the clinical aspect of the cases as observed.

The problem of the treatment of so large a group of cases was of itself a new and difficult one. The physicians of these cases were notified by the State Board of Health of certain centers where clinics would be held, and I made five trips to Vermont, spending two days at a time there, for the purpose of prescribing treatment. Cases were grouped in Burlington, Barton, Montpelier, St. Albans and Rutland, the local hospital in each instance being utilized for the purpose, and to each of these places I went with my senior assistant, Miss W. G. Wright, and investigated and prescribed for the cases by groups. Every possible facility was afforded to me for this investigation, and the work everywhere was made easy and agreeable by the interest and cooperation of the physicians and by the very efficient assistance of the State Board of Health.

The only possible solution of the matter seemed to be in enlisting the family physician and the family to cooperate in the treatment, because to have covered the state by a professional masseuse would have been practically impossible, and it seemed well under the circumstances to make the parents share as much in the responsibility for the treatment of the cases as might be possible. The principle was pursued where possible of seeing the patient with the doctor who was in charge, and after looking the patient over, apparatus was prescribed and provided when necessary, and in all cases that were likely to be benefited by it, in-

structions were given as to muscle training and general routine. A chart was made of each case showing the affected muscles, and this chart was filed with the record of the patient dealing with certain etiologic factors, and deposited with the secretary of the State Board of Health.

It is the purpose of the state board to have these cases seen again in a few months, when it will be possible, by making new charts and comparing them with the original ones, to see how much progress has been made, and at the same time readjust apparatus and prescribe operations.

The whole enterprise is interesting as a piece of constructive medicine, and the results of the treatment will of course be the most interesting part of the investigation, although certain facts already observed seem worthy of analysis.

The epidemic of the summer of 1914 in Vermont was confined almost wholly to the northern half of the state, and there were reported to the State Board of Health 306 cases. Vermont is a state with 355,956 inhabitants, and the occurrence of 306 cases represents a very high incidence of the disease. If one compares it with the New York epidemic of 1907, estimated in the report of the Collective Investigation Committee at 2,000 cases, one finds that in Vermont there were per capita nearly twice as many cases as there were in the New York epidemic. It is of interest in this connection to note that the first large epidemic in this country was reported in the southern part of Vermont by Dr. Caverly* in 1894, when he reported 132 cases. From that time on there has been no serious epidemic in the state. In 1910 the disease began to come more in evidence, and in that year there were 69 cases. There were 27 cases in 1911, only 13 cases in 1912, and 47 cases in 1913. From these yearly fluctuations the jump to more than 300 cases in 1914 is made.†

*Caverly, C. S.: Notes of an Epidemic of Acute Anterior Poliomyelitis, *The Journal A. M. A.*, Jan. 4, 1896, p. 1.

†Caverly, C. S.: Bull. Vermont State Board of Health, June 1, 1914.

There applied for treatment at the clinics 235 cases, but a certain number of cases of other years were brought for advice, and a certain number of cases of other paralyses were brought, cutting down the number of the 1914 cases to 149, and it is from these 1914 cases that the conclusions presented are drawn. The youngest patient observed was a nursing baby aged 6 weeks at the time of the onset, and the oldest patient was 41 years old. One striking feature in the epidemic was that the incidence among older persons was unusually high. Between 10 and 20 there were thirty-eight cases, between 20 and 30 six cases, and between 30 and 40 two cases.

The severity of the infection is notable in this epidemic, inasmuch as the percentage of deaths was in the neighborhood of 17, which is high. For example, in Massachusetts, in 1,599 cases in the years 1907-1910 inclusive, the death rate was 7.9 per cent. Of course the apparent death rate depends on the thoroughness with which cases are reported, but in Vermont there is no reason to believe that cases were overlooked to any large extent, and it is probable that nearly all cases of frank paralysis have been reported, so thorough was the investigation of the state board.

The difficulties attending the diagnosis of the affection were illustrated by some cases that were seen. A boy with a fractured elbow, while the arm was in the splint, was seized with an attack of fever, and had increased pain in the elbow. When the splint was removed, the arm was found entirely paralyzed from the shoulder down, and on examination was found to be a typical case of infantile paralysis. A boy with a congenital deformity of the foot, a talipes equinus, came to the clinic with a history of having always been lame, but after a feverish attack being much lamer. Analysis of the case showed a mixture of congenital deformity and recent infantile paralysis. In one family two children of about the same age were brought,

one with a typical cerebral hemiplegia of three years' duration, the other one with a typical infantile paralysis of an arm and a leg. The family had classed them both as the same condition. Incidentally, two contemporaneous cases seen at the Children's Hospital may be mentioned, one of a child with a congenital dislocation of the hip in one leg and infantile paralysis in the other, and another child with an obstetric paralysis of the arm on the right side existing from birth, and a subsequent infantile paralysis of the leg on the same side. In most cases, however, the diagnosis was easy at the time when the patients were seen, and as a rule had been promptly made by the attending physician.

An attempt was made to see whether any relation existed constantly between the severity of the attack and the degree of the paralysis, because in a paper published some years* ago, the statement was made that, in general, the severity of the attack corresponded to the intensity of the paralysis. The difficulty of finding out from the parents the facts as to the severity of the attack made information of this sort of no great value, because the majority of parents were inclined to regard the attack as serious in any event.

All patients were stripped, and the muscles were individually tested as to function. Cases in babies, which could not be examined in this way, are not included in the report. The muscles were classed as wholly paralyzed, partly paralyzed, and normal. By wholly paralyzed is meant that no response could be elicited from a voluntary attempt to contract the muscle either in a contraction of muscular fibers or tightening of the tendon. For this determination the muscle was given the most favorable condition of leverage; for instance, to straighten the knee when the patient sits with the leg hanging requires a strong quadriceps, because the weight of the leg must be raised from the vertical to the horizontal position. If, however, the patient is laid on the affected side

*Lovett, Robert W., and Lucas, W. P.: Infantile Paralysis, *The Journal A. M. A.*, Nov. 14, 1908, p. 1677.

on a smooth table and the knee flexed, much less muscular power is required to extend it, and very low grades of remaining power can thus be detected. On this basis, if no power in any position could be detected in response to voluntary impulse, the muscle was classed as *wholly paralyzed*. If any degree of contractile power in the muscle or tendon could be detected, or if the muscle had fair but not normal power, it was classed as *partially paralyzed*. Otherwise a muscle was classed as normal.

The condition of each muscle was then marked on charts, which I originally obtained from Dr. E. A. Sharpe of Buffalo, and these charts form the basis of the following analysis. It is not possible to determine with much accuracy the paralysis of the smaller muscles of the shoulder, hip, hand or foot, but with regard to the main muscles of the trunk and limbs, the determination could be made with a fair degree of certainty. The cases under analysis were seen in the winter of 1914-1915, and were of from two to six months' duration when examined. The majority of cases were of three or four months' duration.

NATURE OF THE PARALYSIS

It became evident that partial paralysis was much more common than total. Of 1,452 muscles affected, 416 were totally paralyzed and 1,036 partly, that is, the relation of partial to total paralysis was as 2.5 to 1. The ratio of partial to total paralysis varied in individual muscles, a matter to be discussed later.

A curious phenomenon was several times observed, where part of a muscle was paralyzed and the other part not. This was observed in the deltoid muscle, where the anterior or posterior half might work independently of the other, and once in the pectoralis major, where the sternal and clavicular parts were separated by function.

The predominance of partial over total paralysis is of

importance. The reason for it would seem to lie in the grouping and relation of the nerve cells in the anterior cornua of the cord. These cells lie in longitudinal bundles, which are naturally largest in the cervical and lumbar enlargements.

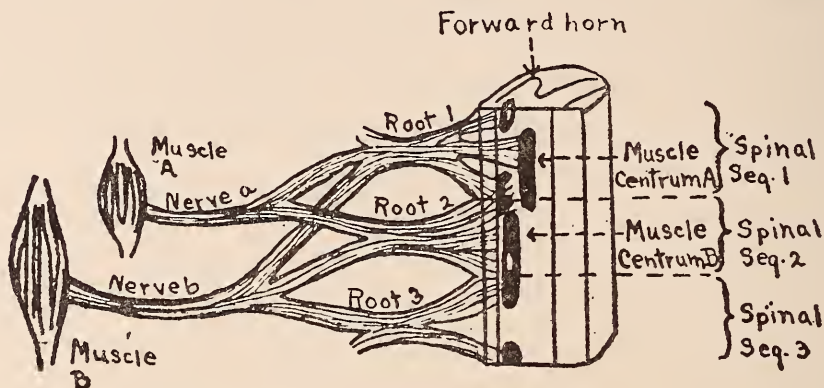


Fig 1.—Radicular and peripheral muscular innervation. Muscle A supplied by segments 1 and 2; muscle B supplied by segments 1, 2 and 3 (Bing).

I quote from Bing:*

Each contains fibers from several anterior roots, and, conversely, each anterior root distributes its fibers among several peripheral nerve trunks. . . . Anterior nerve root lesions, on the other hand, unless very extensive, merely weaken and do not completely paralyze the muscle, owing to the fact that as a rule the muscle is innervated from several roots.

Moreover, we must remember that the poison of infantile paralysis apparently reaches the cord by means of the circulation, and that the main blood supply is from the anterior spinal artery, horizontal branches from which enter the cord at each side at different levels, about 200 in number. The planes of destruction, therefore, are likely to be transverse, while the lines of nerve center association are longitudinal, so that in the case of a muscle which derives its innervation from a group of nerve cells occupying several

*Bing, Robert: *Compendium of Regional Diagnosis in Affections of the Brain and Spinal Cord*, New York, Rebman Company, 1909.

segments, a transverse lesion may well leave certain centers intact, and some power may remain in the muscle. The iliopsoas muscle, for example, is innervated from the twelfth dorsal and first five lumbar segments, the quadriceps from the second, third and fourth lumbar, etc.

This matter of partial paralysis is most important in the matter of treatment, as we shall see when we come to dis-

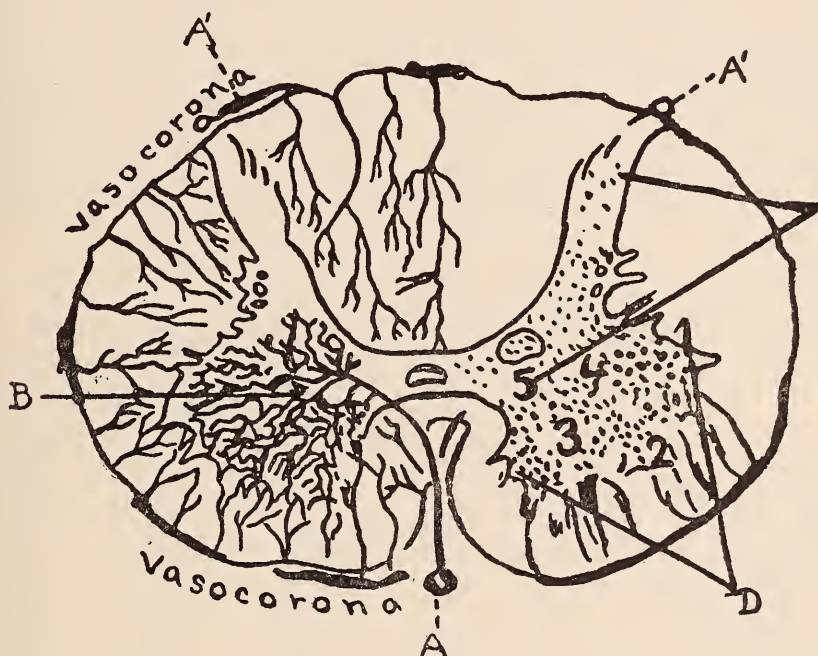


Fig. 2.—Distribution of blood vessels and nerve cells in the transverse section of the cord. *A*, anterior spinal; *A'*, posterior spinal; *B*, area of distribution of sulco-commissural artery; *C*, tract cells; *D*, root cells. 1, postero-lateral; 2, antero-lateral; 3, antero-mesial; 4, central; 5, postero-mesial (Bing).

cuss the therapeutic measure of muscle training, because in such muscles there remains some initiative, and with it the power of developing more muscular volume and new associations by repeated passages of impulses from brain to muscle.

AFFECTION OF INDIVIDUAL MUSCLES

A tabulation was next made as to the affection of individual muscles, which shows that they were affected either partially or totally in the degrees indicated in Table 1. This table gives the number of total paralysees of each muscle, the number of partial and total paralysees, and the proportion of total to partial in each.

The main facts are that the quadriceps, gluteals and gastrocnemius lead in frequency, and that paralysis of leg muscles is much more frequent than of arm muscles. Abdominal paralysis existed in more than half of all the cases (seventy-nine), and affection of the muscles of the spine in more than a quarter (forty). The latter points have a distinct bearing on the occurrence of scoliosis, and indicate, I

TABLE 1.—DEGREE OF AFFECTION OF INDIVIDUAL MUSCLES

<i>Muscle</i>	<i>Number Paralyzed</i>	<i>Number Partial</i>	<i>Number Complete</i>	<i>Proportion of Partial to Total</i>
Adductors	68	52	16	3.2:1
Gluteals	133	106	27	3.9:1
Flexors of hip	81	63	18	3.5:1
Quadriceps	152	119	33	3.6:1
Hamstrings, outer	97	66	31	} 2.7:1
Hamstrings, inner	95	73	22	
Gastrocnemius	128	88	40	2.2:1
Tibialis anticus	119	53	66	0.8:1
Peroneals	96	40	56	0.7:1
Deltoid	57	45	12	3.7:1
Trapezius	49	42	7	6.0:1
Infraspinatus	17	8	9	0.9:1
Pectoralis	29	22	7	3.1:1
Biceps	31	24	7	3.4:1
Triceps	28	22	6	3.6:1
Abdominal	79	64	15	4.3:1
Latissimus dorsi	49	40	9	3.4:1
Spinal	40	36	4	9.0:1
Flexor carpi ulnaris	16	12	4	3.0:1
Flexor carpi radialis	16	11	5	2.2:1
Extensor carpi ulnaris ...	19	14	5	2.8:1
Extensor carpi radialis ...	18	13	5	2.6:1
Opponens pollicis	12	9	3	3.0:1
Extensor pollicis	23	14	9	1.5:1
	<hr/> 1,452	<hr/> 1,036	<hr/> 416	

believe, that such affections are more common than had been supposed. The cases of abdominal paralysis were always symmetrical with two exceptions, one right and one left. This paralysis may occur as the only paralysis in the entire muscular system. When associated with paralysis of other parts, the association is always with leg muscles.

The tibialis anticus and gastrocnemius are the only leg muscles which have been found to be affected by themselves without paralysis occurring elsewhere in the body. Of the former muscle, there were five cases of paralysis, of the latter three. Deltoid paralysis may occur alone in the arm.

The investigation of paralyses of the arm showed (1) that the paralysis was most frequent at the shoulder and diminished in frequency from the shoulder to the hand: (2) that the paralysis was severest (that is, that the percentage of total cases was largest) in the shoulder and diminished as one went toward the hand, and (3) that paralysis of the muscles of the left arm was very much more frequent than of the right arm.

These three factors will be considered and tabulated, and then the leg will be considered in the same respects, after which the possible meaning of these phenomena will be discussed.

TABLE 2.—FIGURES SHOWING THAT PARALYSIS OF THE ARM MUSCLES IS MOST FREQUENT IN THE SHOULDER AND DIMINISHES TOWARD THE HAND

<i>Muscle</i>	<i>Total Paralysis</i>
Deltoid	57
Trapezius	39
Pectoralis	29
Infraspinatus	17
Shoulder group	<hr/> 142 ÷ 4 = 35.5
Biceps	31
Triceps	27
Upper arm group	<hr/> 58 ÷ 2 = 29.0

	<i>Total Paralysis</i>
Flexor carpi ulnaris	16
Flexor carpi radialis	17
Extensor carpi ulnaris	19
Extensor carpi radialis	18
Forearm group	<hr/> 70 ÷ 4 = 17.5
Opponens pollicis	23
Extensor pollicis	12
Hand group	<hr/> 35 ÷ 2 = 17.5

The figures 35.5, 29.0, 17.5 and 17.5 in Table 2 represent the mean paralysis per muscle in each group. The separation at the point where the weight bearing of the arm diminishes is very marked. The ratio between the groups above and below the elbow is 1.8:1.

In Table 3, the last column gives the mean total paralysis per muscle in the region specified. The general ratio in the arm muscles of partial to total paralysis is 2.6:1, or about the same as in all the muscles considered together.

Other facts about paralysis of arm muscles are as follows: In cases in which the muscles of the upper extremity are involved without paralysis occurring at other parts of the body, it is more severe in this region than when the muscles of the legs are also involved; that is, arm paralysis which is strictly regional is more severe than arm paralysis

TABLE 3.—FIGURES SHOWING THAT PARALYSIS OF THE ARM MUSCLES IS SEVEREST AT THE SHOULDER AND DIMINISHES TOWARD THE HAND

<i>Muscle</i>	<i>Total Paralysis</i>
Deltoid	12
Infraspinatus	9
Pectoralis	7
Trapezius	7
Group	<hr/> 35 ÷ 4 = 8.75

	<i>Total Paralysis</i>
Biceps	7
Triceps	6
Group	$13 \div 2 = 6.50$
Flexor carpi ulnaris	4
Flexor carpi radialis	5
Extensor carpi ulnaris	5
Extensor carpi radialis	5
Group	$19 \div 4 = 4.75$
Extensor pollicis	3
Opponens pollicis	9
Group	$12 \div 2 = 6.00$

TABLE 4.—FIGURES SHOWING THAT PARALYSIS OF THE MUSCLES OF THE SHOULDERS, ARM AND FOREARM IS MORE FREQUENT ON THE LEFT SIDE THAN ON THE RIGHT

<i>Muscle</i>	<i>Right</i>	<i>Left</i>
Deltoid	24	33
Trapezius	18	21
Infraspinatus	6	11
Pectoralis	11	18
Biceps	11	20
Triceps	12	15
Flexor carpi ulnaris	7	9
Flexor carpi radialis	7	9
Extensor carpi ulnaris	7	12
Extensor carpi radialis	6	12
Extensor pollicis	5	7
Opponens pollicis	10	13
Total	124	180
Ratio	2	to 3 (very nearly)

which exists in combination with more general paralysis. This statement rests on the analysis of fifty-eight cases.

The relative frequency of paralysis of the thumb muscles (the opponens and extensors) was noticeable, and was found in thirty-five cases (Table 4). It probably existed in more cases among those first examined and was overlooked, because at the beginning of the inquiry it was not realized how commonly this existed in cases in which there was little or no other paralysis of the lower arm.

With regard to paralysis of the muscles of the lower limb, the following facts were observed, which are of importance as contrasted with the similar observations in the arm:

1. The paralysis was on the whole more frequent at the hip, and diminished in frequency toward the foot; that is, the individual muscles in the upper segment were more often affected than in the lower (Table 5).

TABLE 5.—FIGURES SHOWING GREATER FREQUENCY OF PARALYSIS IN UPPER SEGMENT THAN IN LOWER

<i>Muscles</i>	<i>No.</i>
Quadriceps	152
Gluteals	133
Gastrocnemius	128
Tibialis anticus	119
Outer hamstrings	97
Inner hamstrings	95
Peroneals	96
Flexors of hip	81
Adductors	68

2. The paralysis was on the whole lightest in the hip, next lightest in the thigh and severest in the lower leg; that is, the proportion of total to partial paralysis increased as one went away from the hip toward the foot (Table 6).

TABLE 6.—PROPORTION OF PARTIAL TO TOTAL PARALYSIS IN THE MUSCLES OF THE LOWER EXTREMITY

<i>Muscles</i>	<i>Partial to Total</i>
Gluteal	4.0:1
Quadriceps	3.6:1
Flexors of hip	3.5:1
Adductors of hip	3.2:1
Hamstrings	2.7:1
Gastrocnemius	2.2:1
Tibialis anticus	0.8:1
Peroneals	0.7:1

RIGHT AND LEFT LEG

With regard to the relative frequency of paralysis in the right and left leg, the figures show in a total of 954 paralyses of leg muscles that there were 465 muscles paralyzed in left legs and 489 in the right, showing no especial difference in the affection of the two sides. This is a marked contrast to the predominance of left paralyses in the arm.

ANALYSIS OF PREDOMINANCE OF PARALYSIS IN LEFT ARM
AND FREQUENCY OF DISTRIBUTION IN ARM AND LEG

Certain interesting problems are opened up by this study of arm and leg affections which demand analysis. The facts of paralysis occurrence are as follows: The muscles of the limbs nearest the trunk are more frequently affected than the distal ones; the left arm muscles are noticeably more frequently affected than the right. The leg muscles in the right and left leg are equally affected.

The facts of use or function are that the right arm is much more actively used than the left, not only more frequently, but also for more varied and complicated and finer movements; the legs are used equally. It would therefore seem that muscles used actively, continuously and in a complicated way were more likely to escape than those less used, or used for simpler, less continuous work. One would suppose that the blood supply would be more free around the spinal centers where the motor activity was greatest and most complicated, and perhaps less free where the motions were less frequent and complicated. This would account for the predominance of left arm paralysis and the equal paralysis of both legs.

After these figures were worked out, it seemed that such a relation between right and left should appear more in older than in younger patients, because in the younger ones the differentiation between the right and left arms is, of course, less marked than in the older, younger children being much more nearly ambidextrous. If such a relation between right and left arms rested on a functional basis, it would be expected that there would be a larger proportion of left arm paralysees in older than in younger patients. In twenty-four patients 5 years old and younger, there were twelve left arms and twelve right paralyzed, a ratio of 1 : 1. In twenty-seven patients over 5 years of age (from 6 to 38

years) there were twenty cases of left arm paralysis and seven of right, a ratio of 3 : 1.

I should wish to acknowledge here my great indebtedness to Prof. W. B. Cannon and Assistant Professor Martin of the Physiological Department of Harvard University for much assistance on the physiologic side of the problem.

This also accords with the distribution of the paralysis in both arms and legs, which has been shown to be most frequent near the trunk. The demands on the hip and shoulder muscles are simple and less continuous than on the muscles of the lower leg and forearm or of the hand and foot. The latter are continuously active in small, fine, complicated movements, whereas the larger muscles nearest the trunk deal with the coarser and less frequent movements. The relation between the activity of the proximal and distal parts of a limb are not unlike those of the left and right arm in their relative use. It seems probable from these facts and this grouping that, on the whole, muscle centers given to finer, complicated, more frequent movements have a more active blood supply and are less likely to attack on their nerve centers from the virus of infantile paralysis than the centers of muscles functioning in heavier, less complicated and less frequent movements.

ANALYSIS OF SEVERITY DISTRIBUTION IN ARM AND LEG

In the next place, it has been shown that the muscles of the upper extremity are more severely affected nearest the trunk and less severely lower down, whereas in the leg this relation is reversed, and the largest proportion of severe paralysis is in the lower leg and foot. This is estimated on the proportion of total to partial paralysis in the individual muscles.

This puzzling phenomenon is more nearly correlated to the weight coming on each muscle in the activities of the upright position than to any other factor. The great major-

ity of these patients were walking in some form or other, so that the weight-bearing position may fairly be taken into account.

At the shoulder, the deltoid, triceps and biceps all help to hold the arm up against the shoulder joint, and the weight to be met not only in this suspensory function, but also in attempted movements, is greatest at the shoulder and less as one goes down the arm, because the weight of the whole arm is obviously more than the weight of the lower one or two segments. Upper arm muscles, consequently, have more weight to handle than lower arm and hand muscles.

In the leg, on the other hand, the weight to be met in muscular function increases as we go from the hip to the feet, as of course there is greater superincumbent weight at the lower leg than at the hips, so that the lower leg muscles must raise more weight than hip muscles in walking, for instance. There is, of course, no proof that this variation in severity of paralysis is caused by this greater or less weight to be met in muscular function. The explanation accords with the facts, however, better than any other seems to do. Severity distribution cannot be connected with size of muscles, or function of a peculiar sort. It cannot be explained by local circulatory sluggishness affecting dependent parts. It is not associated with anterior or posterior muscles, nor is it easy to connect it with spinal localization. It seems purely a segmental limb distribution, and whether it is or is not the correct explanation, severity of paralysis is proportionate to the weight to be met by the muscles of the different levels, not because this factor influences in any way the original affection of the cells, but because it may retard the recovery of those muscles working against the greatest weight.

This suggestion has a direct bearing on the matter of treatment, for if it is correct it may be interpreted into

showing the ill effects on muscular recovery of overuse, a matter which will be discussed later.

Infantile paralysis has been heretofore regarded as a haphazard affection of muscles, most frequent in the leg, and in the cord lesion it appears to have a purely accidental distribution most marked in the lumbar enlargement. It is possible, however, that there are other factors than the cord lesion which determine the ultimate condition of affected muscles. The analysis just made as to frequency and severity of paralysis shows that in cases some months after the attack there are apparently existing conditions not easily to be explained by the cord lesion alone, but suggesting that function, and especially the function of maintaining the upright position, may have something to do with determining the ultimate distribution.

ASSOCIATED PARALYSIS IN THE LEG

Further pursuit of the inquiry suggested by the analyses just given takes up the question of muscular grouping in the leg. If the paralysis were a purely segmental affair wholly determined by the cord lesions, there would be in the limbs a roughly segmental distribution, muscles at the front and back of the thigh and front and back of the leg being more often paralyzed in this combination than in any combination suggesting associated function. In other words, opponents would be more often paralyzed in combination than would synergistic or functionally associated muscles at different levels.

The leg rather than the arm was selected for this analysis because of the greater simplicity of function in the former. In the arm, rotation movements complicate the more purely forward and backward movements of the lower extremity.

The inquiry then resolved itself into the investigation of whether associated or antagonistic muscles were most often paralyzed in combination in the legs.

The muscles in the leg most closely associated functionally are the gluteals, the quadriceps and the gastrocnemius, for they are the muscles which maintain the upright position. The gastrocnemius holds the tibia upright on the foot, the quadriceps holds the knee straight, and the gluteals hold the trunk erect on the legs. The associations were as follows: If the quadriceps is paralyzed, either the gluteals or the gastrocnemius, or both, are almost always associated with it. In 109 cases there were only two exceptions. In three cases the quadriceps had no association in the leg. In the 109 cases of quadriceps paralysis, to contrast with the 106 associations of gluteals or gastrocnemius, there were only fifty-eight associations of paralysis of one or both hamstrings. The quadriceps, therefore, is affected nearly twice as often with its associated muscles as with its antagonists.

When the gastrocnemius is involved, the quadriceps or gluteals were involved in 108 out of 109 cases; but the antagonists of the gastrocnemius, the extensor longus digitorum and the tibialis anticus, either one or both, were paralyzed in combination with it in only sixty-six cases.

EXPLANATIONS OF PREDOMINANCE OF ASSOCIATION PARALYSIS

This predominance of association paralysis is susceptible of several possible explanations, of which the following may be mentioned:

1. The muscles which maintain the erect position are all very large, and must have large centers composed of many motor cells. On account of their very extent, therefore, they are more likely to be affected than smaller muscles by a generally distributed destructive process in the cord. That this is not altogether acceptable is shown by reference to Table 1, in which it will be seen that the tibialis anticus and peroneals, which are small, are of high incidence, and the pectoralis major, a large muscle, is of low incidence.

2. The second explanation is that associated muscles may be so intimately grouped in the arrangement of their motor centers in the cord that they are more likely to be involved in the same lesion than opposed muscles would be. If, however, one may trust to the present data on the segmental innervation of these muscles, this view is not borne out. The gluteals are credited to the fourth and fifth lumbar and first and second sacral segments, the quadriceps to the second, third and fourth lumbar, and the gastrocnemius to the fourth and fifth lumbar and first and second sacral. The hamstrings belong to the third, fourth and fifth lumbar and first sacral, and the tibialis anticus to the fourth and fifth lumbar. These data are taken from Bing, and do not suggest that the levels of the different motor centers favor the groupings of associated muscles together or antagonists together, nor does the grouping of the bundles as seen in cross section particularly favor this view.

As to the arrangements of the columns in the cord I quote again from Bing:

In general it may be said that the centers . . . for the muscles of the proximal segments of the limbs are to be found in the ventromeseal, while the two lateral groups govern the remaining segments of the extremities. The centers for the coarser movements of flexion and extension are in the neighborhood of the periphery, while those of the finer movements (e.g., of fingers and toes) lie nearer the central groups.

The following statement, from Van Gehuchten and De Buck,* would seem also to be against the assumption that anatomic grouping of centers of associated muscles explains the associated paralyses spoken of:

Extending this conclusion to all the cellular groupings of the anterior horn of the spinal cord, as well in the cervical as in the lumbar enlargement, they defend the idea that the different natural grouping of nerve cells which exist at the

*Van Gehuchten and De Buck: *Rev. neurol.*, 1898.

periphery of the anterior horn in the cervical and in the lumbosacral enlargement are *en rapport*, with neither the peripheral nerves nor isolated muscles, nor with groups of muscles fulfilling the same physiological function. They are uniquely and exclusively *en rapport* with the muscles of the different segments. Medullary motor localization, therefore, is neither nervous nor muscular, it is segmental.

3. The third explanation for the predominance of residual paralysis in associated rather than antagonistic groups may be in the functional relation of the muscles themselves. Three muscles, the gluteal, the quadriceps and the gastrocnemius, work together to maintain the upright position, and if a whole leg is lightly affected, it may be that the association of these muscles in function may retard their recovery by their intimate and necessary functional dependence on each other; especially if one were seriously affected, it might retard the recovery of the muscles associated with it by throwing more work on them than they were able to perform in their affected condition, which condition would not obtain with regard to antagonists.

At this stage of the inquiry, it is not possible fairly to choose either one of these three explanations as the one to be accepted to the exclusion of the others. Perhaps the truth lies in a combination of the assumption that nerve centers of associated muscles are contiguous and that associated function may also be a factor.

CLINICAL APPLICATION

Such are the facts elicited in this preliminary analysis. Of course they mean something, and I have tried to interpret them as fairly as possible and let them tell their own story without reading into them anything that does not belong there. I have proved nothing, and my tentative conclusions are merely suggestions. Nothing more is warranted. But I should like to make what I believe to be a fair application of

these conclusions to certain clinical aspects of treatment, and to leave the reader judge as to their applicability.

I regard one point as fairly well established by these figures, namely that there is another factor beside the plain anatomic distribution of the lesion in the cord which determines something of the extent and severity of the residual paralysis.

It will be necessary to review in a few words what I believe to be the present status of the treatment of infantile paralysis before proceeding to the possible explanation of these facts. The affection is an acute one, and treated in the early stage by rest, because paralysis is generally present in the limbs, making activity impossible, and because the patients are generally sensitive, and movement and handling are painful. It seems rational not to attempt to stimulate by massage or electricity or handling the peripheral connections of such seriously disordered nerve centers, and the best accepted usage is to let such cases alone, only striving to prevent contractions until sensitiveness has disappeared, but to allow the process of repair to go on undisturbed. The statement that electricity will kill the organism in the early stage is of course wholly unreasonable and unproved. It is the custom to prescribe hexamethylenamin at this stage, and many of us are of the opinion that clinically it is of use; but in the Vermont series there was an interesting and suggestive case, in which a child had been taking 5 grains of hexamethylenamin for three or four days before coming down with a typical attack of infantile paralysis.

With regard to holding off massage, etc., until after sensitiveness has disappeared, it rests on theoretical considerations and on empiric grounds. I had been convinced that it was correct, but thinking that perhaps I had fallen too much into a rut, after consultation last winter with a colleague skilled in neurology I attempted to institute earlier than usual the therapeutic measures in a case still tender,

in which the sensitiveness seemed of unreasonably long duration.

A boy of 5½ years, paralyzed in Vermont in August, 1914, was under my care at the Children's Hospital. At the end of nine weeks he was still decidedly sensitive in the paralyzed limbs, and on October 22 I decided to try the gentlest massage of five minutes a day to each leg. The massage was given by a highly skilled masseuse of great experience under my personal direction. In one week the boy was so sensitive that a cradle had to be put on the bed to protect him from the pressure of the bedclothes, and massage was omitted and both legs were put up in plaster of Paris. In one week more the plasters were removed, and it was found that the sensitiveness had wholly disappeared, nor did it ever return. One case, of course, proves nothing; but it is, I believe, indicative of a general principle.

The tenderness may last from two to three months after the attack, and a perfectly inactive treatment is hard to pursue when the family has heard of the wonders of electricity and massage, and is anxious not to lose time. But so long as the tenderness lasts, the best practice is to let the patient alone so far as active treatment goes. Frequent changes of position are desirable, and there is no objection to the sitting position for the convalescent, to outdoor air, or to immersion in a warm bath with whatever active movement under water may be accomplished without discomfort.

There is no danger that the joints will stiffen, and in the first weeks the only troublesome complication to be feared, as has been said, is contraction of the Achilles tendon.

With the disappearance of tenderness, the time for active treatment has begun, and the sooner the patient is put on his feet and resumes activity the better. It seems probable on general principles that in cases of any degree of severity, even if tenderness disappears earlier than four weeks, active treatment should not be begun before that time. The general condition of the patient must not be neglected, as many of the children at this time have not wholly

recovered from the effects of infection, and are anemic, poorly, and easily fatigued.

The therapeutic measures at our disposal in fairly early cases are massage, electricity and muscle training.

Massage may be expected to improve the local and general circulation, to facilitate the flow of lymph, and to retard muscular deterioration. It cannot, however, be expected to facilitate the transmission of a motor impulse from the brain to the muscle.

Electricity is less highly regarded in the treatment than was formerly the case. The unintelligent use of electricity month after month to the exclusion of other measures has been one of the handicaps which has stood in the way of the best progress in many cases. It is quite possible that it may improve the muscular condition. Statements of its value rest as a rule on bare personal assertion or on the unusually rapid improvement of individual cases; but cases vary greatly in their rate of improvement, and the only way to judge of the value of electricity is to use it on one side of the body in bilateral cases and use the other side as a control. In the winter of 1913-1914, some cases in private practice were given daily treatments of galvanic electricity on one side and none on the other, while daily muscle training was being given by my assistant, who was not told which side was receiving the electrical treatment. At the end of some months she was asked if either side had shown more rapid improvement than the other, and no difference had been noted. This simply confirmed my general experience of many years of less careful observation.

Muscle training, on the other hand, rests on a sound physiologic basis, works out empirically better than any other of the measures, and the large proportion of partial paralysis in the cases observed shows its reasonableness. It consists in an attempt to induce a voluntary impulse to pass from the brain, down the motor tracts of the upper neuron,

through the appropriate centers to the selected muscle. By the disease, certain spinal motor centers were destroyed, and can therefore no longer act to distribute motor impulses to their muscles. But such spinal centers and their connections are complicated affairs, and every muscle is connected with several centers, every center sends impulses to more than one muscle, and, moreover, the connections between the spinal centers are many. Unless, therefore, the destruction in the cord has been a very extensive one, it is likely that some of the motor centers in any one region will have escaped destruction, and that it may be possible to establish new connections around the destroyed centers. If a railway wreck occurs in the main line and the track is blocked, it is often possible to send trains by means of a branch line around the obstruction, so that service between the terminals is maintained. In the same way, after a wreck of certain nerve centers, it may be possible by a modified route to send a motor impulse from brain to muscle. On this principle of establishing new connections and opening new paths rests most of the claim of muscle training.

Muscle training in its most obvious form consists in aiding the patient to perform a certain movement with the hope of stimulating an impulse from the brain to the muscles. If, for instance, the dorsal flexors of the foot do not act, through being stretched, weakened, partly paralyzed, or wholly paralyzed, in the exercise the foot is dorsally flexed with the hand and the patient directed to assist. If there is any muscular response, less and less aid is given to the muscle by the hand, and it may be that in this way it can be trained to perform its function.*

Another and equally useful form of muscle training consists in getting the patient on his feet at the earliest possible moment in order to call forth the instinctive muscular actions induced by the efforts and balance. Even before it

*Wright: Muscle Training in the Treatment of Infantile Paralysis, Boston Med. and Surg. Jour., Oct. 24, 1912.

is possible to make much progress in this way, sitting is useful for the spinal and trunk muscles.

THE USE OF APPARATUS AND BRACES

Many patients at the beginning are unable to stand without apparatus, because, for example, the knees flex on account of weakness or paralysis of the quadriceps muscle. In these cases a caliper splint should be applied to hold the knees straight. If the feet roll in or out, varus or valgus braces should be applied. If the spine or abdomen is involved, a corset or jacket should be worn. Crutches are at first necessary in cases of paralysis of both legs. In other words, if the standing position induces malposition, such malposition must be corrected, because nothing but harm can come of it.

The fear that the early use of apparatus will promote muscular atrophy is wholly unreasonable, because disuse is bad. Braces should mean the upright position, and the upright position means more muscular activity. The best way to avoid wearing a brace permanently is to put it on early and keep it on as long as necessary. For a growing child to walk about with a malposition is to bid for a permanent deformity.

DEFORMITY

If fixed deformity exists, it must be removed before treatment of any sort can be satisfactory. By fixed deformity is meant a condition in which the functions of a joint are limited, in which its arc is restricted.

An analysis of the deformity of these cases seen from two to six months after the onset was as follows:

Equinus was the most frequent deformity, and was present in sixteen cases. It seems to me very creditable to the medical profession of Vermont that the proportion of equinus should have been so small.

There were nine cases of flexion of the knees and seven of flexion of the hips, sixteen cases of scoliosis, and four cases of hyperextension of the knees, one case of torticollis, and two cases of calcaneus deformity due to gastrocnemius paralysis.

The grade of contraction deformity in these cases was a surprise to me, as I had not realized that in so short a time such serious deformities could arise. There were cases of scoliosis of only a few months' duration which I should have supposed it would take at least one or two years to acquire. The flexion deformity of the hips and knees in one or two instances was striking, in one case one knee being flexed to 90 degrees and the other to 45, with both hips in flexion contraction. Other cases of severe contraction of the knee and hip, only to be remedied by stretching or tenotomy, were seen among the 1914 cases. Contraction of the shoulder in a forward position, not allowing the outward, upward and backward movement, was found in three cases of deltoid paralysis.

The operative attack on deformity, apart from the performance of minor tenotomies and operative measures in general, are by common consent to be postponed until three years from the acute attack. Operative treatment will not be considered in this paper because it is by far the best formulated part of the treatment, and because lack of time forbids.

OVERFATIGUE AND OVERUSE

We come now to the final, and what I believe to be the most important part of the paper, namely, the possible effect of overfatigue and the overuse of massage on returning muscular function, a phase of the treatment question almost wholly neglected.

If we take the case of a partly paralyzed muscle with some remaining power, we are anxious to bring about in

that muscle the greatest possible return of functional power; that is plainly our object of treatment. Now the rational exercise of a normal muscle results in increase of size and power of that muscle, and presumably the result would be the same in a muscle weakened by infantile paralysis which was rationally and physiologically exercised or massaged. We are, however, dealing with muscles in many instances very weak and incapable of doing much work, and it must be an easy thing to overexercise them.

As to the question of this overuse, the following facts are suggestive: The majority of the early cases seen in Vermont showed partial rather than total paralysis; the gastrocnemius muscle in cases of early paralysis was quite different from the stretched, lengthened and powerless gastrocnemius muscle of late cases. Muscles partly paralyzed in which power is returning may be rendered functionless by slight grades of overuse. These considerations all have a bearing, and in connection with observations to be mentioned, indicate the possibility that in many cases of infantile paralysis we are encouraging in partly paralyzed muscles a function wholly beyond their ability, and are thus delaying their return of power and possibly converting partial into total paralysis. The observations follow:

It has been repeatedly observed in my private practice that power might begin to return in a very faint degree to a muscle while under muscle training, and that with care this power would steadily increase, but if that muscle were exercised even very gently every day, that power would diminish or disappear, so that we exercise such muscles only once in three days at the outset, increasing the work most carefully.

A young man under my care, severely paralyzed in both legs, six months after the attack showed some return of power in the peroneals. This developed and was exercised in the usual way, but he was so delighted with the new

function that one year after the attack he tried it at intervals all of one day on the principle that if a little exercise were good a large amount would be better, and the power promptly disappeared, never to return in full amount after some five months.

A young man with a paralysis of the left arm acquired in Vermont in September, 1914, was brought from New York to see me in December, 1914. He was having massage and exercises daily from an apparently competent masseur, and was urged to use his arm as much as he could to stimulate returning function; but for a month he had not improved, and the parents therefore decided to send him to me for treatment. He could not, however, come to Boston for a month, and asked what treatment he should pursue in the meantime. A sling supporting the shoulder was put on, he was forbidden to use the arm except at meals, and massage was stopped. After a month of this routine he showed at least 25 per cent of increase of power.

I saw with Dr. F. B. Percy of Brookline, Oct. 19, 1914, a child of 10 with a total paralysis of the anterior tibial muscle and partial paralysis of the gastrocnemius. Ten days after the attack, sensitiveness had gone, but the child could only walk badly and unsteadily. She was kept quiet for a month more, when she was allowed to walk a few steps daily. She made a remarkable gain, and massage and muscle training were begun, Jan. 6, 1915, although the amount of walking was not increased to any extent, the child walking only a very little. In four months the gastrocnemius and tibialis anticus had apparently nearly normal power when their resistance was tested by the hand, but the child still limped a little. Feb. 24, 1915, four months after the attack, the mother was asked to keep this child practically off of her feet for two weeks while the other conditions of treatment were the same. At the end of this time the limp had disappeared.

Oct 5, 1914, I saw a child of 5 with nearly complete paralysis of one leg below the knee from an attack three months previous. The child walked badly, but was much helped by a brace to hold the foot at right angles to the leg. Muscle training was started, and the child improved satisfactorily, evidences of returning muscle power becoming plain. March 6, 1915, the mother made a statement that attracted my attention, namely, that the child walked better in the morning than at night. She was asked to keep the child off of his feet as much as possible for a month,

restricting walking to the greatest possible degree. April 6, 1915, examination showed during the month a very striking increase in power in the muscles controlling the foot, and it was evident that the progress in the last month had been far greater than in any previous month, and the child walked as well at night as in the morning.

These illustrative cases seem to me to show that much smaller degrees of overuse may be deleterious than is generally supposed. Probably any of us would agree that gross and persistent overuse of partly paralyzed muscles would be undesirable; but it seems to me reasonable that in the early stage of returning power, we should be exceedingly careful in the use of muscles in walking and in the use of heavy and prolonged massage, much more careful than we are at present, if I may judge the practice of others by my own previous methods.

I hesitate to reason from an unproved conclusion in this connection; but may I once more call attention to the fact that the proportion of total to partial paralysis is greatest in the muscles which have the greatest weight to oppose in the standing and walking position and least in those which have the least weight, in a series of cases observed some months after the acute attack. If overuse is the harmful factor that I believe it to be in retarding recovery, its effect would be noted in just those muscles which show the highest proportion of total paralysis.

CONCLUSION

It would be useless to present a summary of a paper which is only a summary in itself. It is merely the preliminary report of a series of observations bringing out certain facts apparently bearing on some of the phenomena of the disease. Certain of these facts may later prove of practical significance or they may not. One of the observations I believe to bear on the question of treatment, but even of that

one cannot be sure until after the second series of observations is made on these same cases. This may perhaps throw some light on the later condition of the muscles—light which may have some practical bearing.

234 Marlborough Street.

INFANTILE PARALYSIS IN VERMONT

A REPORT OF THE PROGRESS OF CASES BETWEEN
JANUARY, 1915, AND JULY, 1915.*

ROBERT W. LOVETT, M.D.

AND

ERNEST G. MARTIN, PH.D.

of Harvard University

IN December, 1914, the State Board of Health of Vermont, through the generosity of an interested citizen of the state, was enabled to undertake the treatment of the cases of infantile paralysis occurring in the summer of 1914, and the matter of treatment was undertaken by one of the writers, the general aspects of the epidemic and the epidemiology being undertaken by the Rockefeller Institute of New York. Five trips were made to Vermont for the purpose of seeing and prescribing for cases. Clinics were held at Barton, Burlington, Montpelier, St. Albans and Rutland, the cases being assembled in these places for the purpose of examinations. There applied for treatment at the clinics 235 cases, but there were a certain number of cases from other years, which cut down the number of the 1914 cases applying for treatment to 149.

In each of the cases an examination was made, and the muscles affected were marked on a chart as "Totally paralyzed," "Partially paralyzed" or "Normal." The needs of the case were then formulated, braces were prescribed when it seemed necessary, certain minor operations were advised, and in cases where it seemed likely to be of use the parents were instructed in muscular exercises to be given to the

*Read at the Annual Meeting of the Vermont State Medical Society, Oct. 14, 1915.

patients for the development of partly affected muscles. Certain cases were not available for this treatment, because they were either too severely affected or too young, but in the majority of cases this treatment by muscular exercises was instituted, and the parents were urged to follow it up.

Subsequently these 149 cases were analyzed as to various points of interest with relation to the relative frequency of muscles affected, and other points likely to be of practical importance, and these conclusions were published in the *Journal of the American Medical Ass'n*, June 26, 1915.

In order to carry the work through, at the request of the Board of Health, a second series of clinics was held in Vermont by both of the writers in July, 1915, and cases were seen at the same places as in January. On this trip there was used a newly devised method of testing the muscular strength of affected muscular groups. It had proved so useful in a preliminary trial at the Children's Hospital, Boston, in the spring and early summer of 1915 that it was deemed worth while to attempt in Vermont to secure more definite information than could be obtained by the usual methods of examination as to certain phenomena of the disease which it was believed would be of value in regard to problems of treatment. Muscle charts were also made on the second trip, but in contrast with the closer method of examination devised they proved to be of much less value than the figures from the new method.

One of the most important facts developed by the original examination of the Vermont cases was that in something more than a third of the 149 cases examined on the first trip partial paralysis was more common than total paralysis, a fact which apparently had not before been realized. Now the term "partial" paralysis indicates any condition from that of a muscle which seems perhaps a little less vigorous than normal to one which shows only a flicker of contraction on attempted voluntary movement. Yet neither of these muscles are normal nor wholly paralyzed.

With regard to the completely paralyzed muscles and the normal muscles there was no special difficulty in classification, but it became evident very early in the investigation that partially paralyzed muscles might gain very much in power without passing out of the class of partially paralyzed muscles, so that a comparison of the charts of the first and second trips in many cases showed the same markings, whereas very great increase in functional activity in many of these cases had occurred. It was evident that any closer study of importance must be made by means of the muscle testing method rather than by the muscle charts, where the power of voluntary contraction was estimated by hand. The study of the progress of the cases by this method is now being carried on, and will be reported on later, and for the present it is possible to speak only of the gross phenomena manifested by the comparison of the records, examinations and charts of the two visits, reinforced by certain preliminary conclusions drawn from the method of muscle testing.

On the second trip, at the five clinics held, 145 cases were seen, examined and prescribed for. Seven of these cases proved to be for affections other than infantile paralysis, reducing the number for analysis to 138. Three of these cases had occurred in the summer of 1915, 96 in the summer of 1914, and the remainder were distributed between previous years, reaching as far back as 1894. Of these 138 cases, 62 were seen for the first time in July and 76 were seen at the time of the visit in the winter and in July also.

The following table shows the number of cases seen at the different clinics:

	<i>Total Cases Seen in July</i>	<i>Cases Seen First Time</i>	<i>Cases Seen 2nd Time</i>	<i>Not In- fantile</i>
Montpelier	22	10	8	4
Burlington	48	22	24	2
St. Albans	30	9	20	1
Barton	21	7	14	0
Rutland	24	14	10	0
Total	145	62	76	7

On the July trip it was found that there had been improvement in every case which had previously been seen with four exceptions. One boy, slightly affected, was said by his parents to be no better, and three patients had not followed the treatment prescribed and had not improved. The improvement in general was partly spontaneous and partly due apparently to treatment. The treatment was muscle training carried out at home, with in most instances restriction of activity. It developed early in the first visit that fatigue was evidently an unfavorable factor, and that exercise and activity must in many instances be cut down.

Before proceeding to any conclusions it may be well to speak of the cases seen at the various clinics somewhat in detail.

Montpelier.—Of 22 cases seen, four were not infantile paralysis, 10 were seen for the first time and eight for the second time. Of the eight cases seen for the second time two walked with braces who had not walked before, one walked without a crutch, one had practically recovered at the first visit and had remained well, and four showed very striking muscular improvement, as shown by comparison of the charts, several deltoids, forearm, gluteals and quadriceps muscles having changed either from total to partial paralysis or from partial paralysis to recovery.

Rutland.—Twenty-four cases were seen, 14 of them being seen for the first time in July. Of the 10 cases seen on both visits five were cases affected in 1911, and may be mentioned separately. Of the 1911 cases one case had been operated upon and was greatly improved. In one there was no note of function, although the parents described the case as better. In one there was no treatment and no improvement, and in two great improvement. Of cases affected in 1914, three showed great improvement, one was too young for an accurate record but was better, and one was better but there was no definite note of function.

Barton.—Twenty-one cases were seen, seven for the first time and 14 for the second time. These comprised some of the severest cases in the state, and improvement had occurred in all. Practical recovery had occurred in two, one could walk who could not walk before, several walked much better, and from a study of the charts it was found that there were many instances of total recovery of muscles partly paralyzed in the winter.

St. Albans.—There were seen 30 cases, nine new ones and 21 seen for a second time. One case of gastrocnemius paralysis had had no treatment and had grown worse from neglect. High heels had been prescribed, which had not been used, and no exercises had been done. The gastrocnemius had stretched and had lost power. This was the only case of gastrocnemius paralysis seen in the state which had not improved under the treatment prescribed, which consisted in elevation of the heels, restricted use and exercises. Three cases were babies too young for exercise, and one case was too unruly for examination. All had improved somewhat. Two who could not walk in January could walk with a brace in July, 13 were much improved, in one the diagnosis was uncertain and the case was probably not infantile paralysis, and in one the notes were unsatisfactory.

Burlington.—Forty-eight cases were seen. Two cases were not infantile paralysis, 22 cases were seen for the first time and 24 for the second time. Of these 24 cases one had had no treatment and showed no improvement, one was too young for accurate data but had improved, one was unruly and could not be carefully examined. In the remaining 21, improvement from slight to very great was recorded. In individual muscles the following changes were noted between January and July.

	<i>Partial Paralysis to Recovery</i>	<i>Total to Partial Paralysis</i>
Deltoid	6	1
Pectoral	3	0

	<i>Partial Paralysis to Recovery</i>	<i>Total to Partial Paralysis</i>
Trapezius	3	1
Triceps	1	0
Arm muscles	2	0
Forearm muscles	1	0
Back muscles	5	0
Abdominal muscles	5	1
Quadriceps	1	2
Hamstrings	2	0
Gastrocnemius	4	0

The two most striking cases in the Vermont series were to be found in the Burlington group. A man of 38, attacked in August, 1914, was helpless and brought to the January clinic on a stretcher, and was with one exception the severest case seen in Vermont. In July he was earning his living by selling farm machinery in his wagon, taking a boy to help him in and out. He walked with one crutch with assistance also on one side, he was able to fish, and had been on a successful fishing trip during the summer. A girl of 18, affected in 1914, in January could barely walk alone. In July she walked with a slight limp. Eight important muscles had recovered under persistent exercise, and the improvement began a few days after starting on exercises, previous to which the patient's progress had been stationary.

In view of the facts developed on the second trip, the State Board of Health determined to pursue the treatment more accurately, it having evidently demonstrated that it was of use, and Miss J. B. Merrill stayed in Vermont from July until the end of September for the purpose of giving the treatment in various parts of the state to patients who were likely to be benefited by it. Two subsequent trips to Vermont were made by Dr. Martin for the purpose of testing the muscles of the cases under treatment, and the improvement recorded was so striking that the Board decided to have Miss Merrill make trips through the winter at intervals of two weeks to various parts of the state for the

purpose of seeing that the treatment was carried out, seeing the parents at frequent intervals, changing the exercises when necessary, and encouraging the parents to a steady pursuance of the treatment.

The advantage of having a quantitative test for muscular strength is that it eliminates personal opinion as to the progress of an individual case and formulates the matter in pounds and ounces, so that by a comparison of the records of the same cases at intervals it is possible to find out whether or not the child is gaining or losing. Although at present it is not possible to present the full data with regard to the results of these observations by the muscle test, it may be said that the cases analyzed so far show that the expectation of improvement in a given muscular group affected but not totally paralyzed by the disease under constant treatment of muscle training from an expert is greater than under supervised home exercises, and that this is more likely to help than home exercises prescribed but given without supervision. Untreated affected muscles in these patients improve least, but even these show an improvement ratio of 1.9 to one, which latter is to be counted partly as spontaneous improvement, but it must be remembered that other muscles in the same case were being treated.

These cases were nearly all affected in 1914, and represent cases at the end of the first year. That spontaneous improvement is so marked at the end of a year is apparently a new point, and one of great importance to us in forming our prognosis. These facts may well give us courage to undertake a more vigorous treatment in old cases, knowing that at the end of a year spontaneous improvement is by no means at an end. Perhaps we may find that it extends much later than this when our observations have covered a longer time. The possibility of improvement in muscles totally paralyzed at the end of one year was shown by observation with the muscle test.

These figures and these clinical conclusions would seem to indicate that the venture of the State Board of Health of Vermont in undertaking on a large scale the treatment of cases of infantile paralysis by modern methods had been successful. That the cases on the whole improved more than would have been expected from spontaneous improvement alone seemed evident. That very great improvement had resulted in certain cases was equally plain, and the fact that so few cases out of the large number treated had failed to improve is apparently encouraging. The cases on the whole were not what one would select as a class on which to demonstrate the efficacy of any treatment. In a measure the patients were the children of farmers living in the country, and often unable to receive much care from the mothers of large families. The parents have nevertheless been sufficiently interested in the treatment as a whole to give it an intelligent trial, with results which apparently have been beneficial.

The care of a large group of cases of infantile paralysis has apparently not been undertaken in this country before, and it presented many difficult problems, but the outcome seems to have justified the undertaking, and it is hoped that when the data of the muscle tests have been formulated some definite increase to our knowledge of infantile paralysis may have been obtained.

CERTAIN ASPECTS OF INFANTILE PARALYSIS

WITH A DESCRIPTION OF A METHOD OF MUSCLE
TESTING*

ROBERT W. LOVETT, M.D.

AND

E. G. MARTIN, PH.D.

THIS paper contains the account of a combined physiologic and orthopedic study of certain phenomena of infantile paralysis. The whole matter owes its inception and present status to the State Board of Health of Vermont, which by the generosity of an anonymous donor was enabled to finance a scheme for the study and treatment of the disease quite unprecedented in its scope and thoroughness. The entire work has been conducted under the direction of the board, which has borne the whole expense of the studies in Boston and Vermont.

The inquiry was started in the late autumn of 1914, the Rockefeller Institute through Dr. Simon Flexner taking charge of the epidemiologic end of the inquiry and opening a laboratory in Burlington, while to one of us (R. W. L.) was assigned the therapeutic side of the problem. Later, for reasons to be stated, the physiologic department of Harvard University was asked to assist in the study, and a system of muscle measurement was devised by one of us (E. G. M.).

This system of measurement was put in use in the early summer of 1915 in the orthopedic department of the Children's Hospital, and later used by us in a trip to Vermont

*A Report to the State Board of Health of Vermont. Copyright, 1916, Am. Med. Assn. and reprinted by permission from the Journ. of the A. M. A., Mar. 4, 1916, Vol. LXVI, pp. 729-733.

made in July, 1915, for the purposes of treatment. So much new light was thrown by its use on certain phenomena of the disease that two subsequent trips to Vermont were made by one of us (E. G. M.) in August and September to make observations on patients under treatment.

The material available for study is as follows: At the first series of Vermont clinics in January and February, 1915, 235 patients applied for treatment, and at the second series in July, 145. From these figures should be deducted seventy-six seen at both clinics, making 304 patients seen and prescribed for in Vermont. One hundred and one patients from the orthopedic clinic of the Children's Hospital, Boston, and from the private practice of one of us have been added to the Vermont material for purposes of study. The results of the analysis of cases on the first trip have been published,* and a short preliminary report on the method of muscle testing has been made.†

In the present paper we first discuss the reasons for the use of a muscle test, second, describe the muscle test, and third, present conclusions obtained by the use of the muscle test in the cases mentioned.

The general thesis of this paper is as follows: The observations in about 300 cases in Vermont have shown that in all cases, old and recent, infantile paralysis has been found in most muscular groups not to be a paralysis in the sense of a complete loss of power, but a weakening of these muscles. Now weakened muscles can, as a rule, be made stronger by judicious exercise, just as can normal muscles. Our therapeutic problem, therefore, requires the closest possible study of such muscles and of the therapeutic means by which their individual power may be increased. It is a very important matter to the parents of a child with a gastrocnemius muscle reduced to only 20 per cent of its normal

*Lovett, R. W.: The Treatment of Infantile Paralysis: Preliminary Report, Based on a Study of the Vermont Epidemic of 1914, *The Journal A. M. A.*, June 26, 1915, p. 2118.

†Martin, E. G., and Lovett, R. W.: A Method of Testing Muscular Strength in Infantile Paralysis, *The Journal A. M. A.*, Oct. 30, 1915, p. 1512.

power whether this muscle ultimately returns to 40 per cent or 90 per cent of its normal strength.

WHY A MUSCLE TEST WAS DESIRABLE

The original study of the Vermont cases, in which the hand was used to estimate voluntary contractile power in the individual muscles, showed in 150 cases of a few months' duration that partial paralysis was more common than total in the proportion of 2.5:1 (1,036 partial to 416 total paralyzes). In the eighty-six cases of the 150 available in July for closer study by means of the muscle test, there were found by this method 111 totally paralyzed and 958 partially paralyzed muscle groups, a proportion of about 9:1. It is evident, therefore, that the bulk of any study will be in cases of "partial paralysis."

At the outset of the Vermont work, muscles were classed as normal, partly paralyzed or totally paralyzed. The first and the last class were clear enough, but in the "partly paralyzed" division we had to group muscles which were just short of normal and muscles which showed only a flicker of movement on attempted contraction. Between these two widely separated conditions existed every degree of disability, yet one must put them all in one class. Exact study of the phenomena under these conditions was impossible.

Again, in the matter of treatment some scale of measuring improvement or the reverse was urgently needed. "Impressions" that electricity of one kind or another, or rest or exercises were beneficial have filled literature; unsupported assertions, marvelous cures, fantastic treatments have too often been advanced on the slenderest of grounds. With the realization that partial paralysis was the usual form of affection, it became imperative to have some scale by which to work out what should be a precise and improved treatment. The muscle test offers a practical quantitative scale

by which the effects of modifications of treatment may be studied week by week and month by month.

The muscle test devised consists of estimating strength of the various muscular groups by means of spring balances and is considered in the next article (p. 252).

CONCLUSIONS DRAWN FROM THE USE OF THE MUSCLE TEST

The following observations rest on studies on 177 patients, on whom 13,000 observations in 400 series were made, forty-four different muscular groups, twenty-two on each side of the body, being available for study.

Distribution of Paralysis. The muscular affection is much more widespread than ordinarily supposed, thus corresponding closely to the pathology as observed of late years. It is rare to find one muscular group affected without manifestations elsewhere; for example, we often class a case as gastrocnemius paralysis, but rarely in our observations (two in 177) has this been found to exist alone, other muscles nearly always being weakened. When one leg is involved it is common to find some weakening of some of the muscles of the other leg. When both legs are affected, some arm muscles are often involved. This fact is well to bear in mind in formulating treatment.

In thirty-two cases of the series, taken consecutively from the files for this analysis, the following data were observed:

Ten cases by manual examination had been classed as having only one leg affected; by the muscle test nine of these showed weakness of muscular groups in the other leg. Of the other twenty-two cases, four had obvious paralysis of all four members. In the remaining eighteen cases of paralysis of more than one limb, no new paralysis was revealed by the muscle test in eight, while in ten, unsuspected involvement in another limb was found.

Distribution of Total and Partial Paralysis. Table 1 shows that there is a predilection on the part of total pa-

ralysis for the lower leg. In the Boston series the cases were of longer duration, of a severer type, many requiring operation, but the ratio is not widely different in the two classes. This confirms the observations made in the 150 cases seen on the first trip, where the peroneal and anterior tibial muscles showed of all muscles the largest incidence of total paralysis.

Partial paralysis may be classed as severe (one third or less of normal power) or moderate (one third or more of normal power). Two classes of cases are available for this analysis, one the Vermont group, mostly affected in 1914, and the other the Boston group, mostly affected before 1912, being largely hospital cases awaiting operation.

The observations given in Table 2 extend the findings of the first study of the Vermont cases, that paralysis is severest in the foot and diminishes toward the hip, and is severest in the shoulder and diminishes toward the hand. In that paper, however, one dealt with the relation of total to partial; here another class is available, and the ratio of severe to moderate bears out the same fact in two widely different classes of cases.

Paralyzed Muscles Should not Lose Power. In the period covered by our observations (two months in the Vermont cases and longer in some of the Boston cases), it has appeared that the strength of affected muscles tends on the whole to remain stationary or to improve, and that a loss of strength is not to be expected, and when it occurs should be investigated. This statement rests on the study of forty-six cases of from one to five years' duration (three from 1911, one from 1913, forty-two from 1914) tested at an interval of two months or more; 549 affected muscular groups were studied, 356 being under treatment by muscle training, and 193 not under treatment. Seven per cent of the muscles under treatment showed a loss of power in this interval (twenty-five muscle groups in thirteen cases). In nearly all

TABLE 1.—MUSCULAR GROUPS SHOWING TOTAL PARALYSIS

	<i>Vermont</i>	<i>Boston</i>
Below knee	74 (82 per cent)	87 (82 per cent)
Elsewhere	16	19
Total	90	106

instances this loss was to be accounted for by conditions known at the time of observation, such as fatigue, ill health, overuse, etc. In the muscles not under treatment there was some loss of power in 24 per cent (forty-seven muscle groups in twenty-one cases). In a large proportion of these, the conditions were such as to explain this observation.

Overfatigue and its Detrimental Effects. The data as to the effect of muscular fatigue from overuse, overexercise therapeutically given and overmassage appeared to be important, and certain clinical observations were formulated after the first series of clinics. A quotation follows:

It has been repeatedly observed in my private practice that power might begin to return in a very faint degree to a muscle while under muscle training, and that with care this power would steadily increase, but if that muscle were exercised even very gently every day, that power would diminish or disappear, so that we exercise such muscles only once in three days at the outset, increasing the work most carefully. . . .

Illustrative cases seem to me to show that much smaller degrees of overuse may be deleterious than is generally supposed. Probably any of us would agree that gross and persistent overuse of partly paralyzed muscles would be undesirable; but it seems to me reasonable that in the early stage

TABLE 2.—DISTRIBUTION OF MUSCULAR GROUPS SHOWING PARTIAL PARALYSIS

	<i>Vermont</i>		<i>Boston</i>	
	<i>Severe</i>	<i>Moderate</i>	<i>Severe</i>	<i>Moderate</i>
Lower extremity:				
Leg	115	109	203	161
Thigh	89	61	101	76
Hip	108	177	117	168
Upper extremity:				
Shoulder and arm	91	87	58	80
Forearm and hand	58	89	18	66

of returning power, we should be exceedingly careful in the use of muscles in walking and in the use of heavy and prolonged massage, much more careful than we are at present, if I may judge the practice of others by my own previous methods.

This matter was confirmed and made more definite by the muscle test. Illustrative cases follow:

Case 1.—A boy of 5, with the onset in 1914, was having at the time of the first test one and one-half hours of massage and one hour of muscle training daily. This was changed, and he was given one hour only of gentle muscular exercise, and the second observation showed a substantial gain of strength in all affected muscles, an illustration of the effect of too much treatment.

Case 2.—A boy with the onset in 1914, in July showed partial paralysis of the left arm. He then began to milk ten cows daily, and in a month had lost strength in all of his left arm muscles except the biceps and triceps, which had gained slightly. The loss was greatest in the muscles of the forearm, which were of course those most exercised in milking, an illustration of the effect of overuse of the muscles as a cause of loss of power in the overused muscles.

In this matter of the study of the detrimental effect of fatigue and the beneficial effect of proper muscular exercise lies apparently one of the most important uses of the method. We need to know the dose of the remedy which we are using.

Variations in General Muscular Strength. Children are subject to variations in muscular strength at different times, the whole scale of readings for both normal and affected muscles being sometimes decidedly higher or lower than their usual values; but the raising or lowering of the whole scale while the relative strength of the muscles remains practically the same is apparently dependent on general conditions and does not affect the question of paralysis. During the great heat of September, 1915, a decided falling off in muscular strength was noted in several cases whose

normal scale was known, and observations were abandoned until it became cooler.

Abortive Paralysis. That cases of so-called abortive paralysis are often really cases in which paralysis exists, but is too slight to be detected by the ordinary examination, seems likely. Two observations bear on this point:

In one family in Burlington, three children were similarly affected at about the same time. One showed a frank paralysis, and the other two did not, and were classed as abortive cases and showed nothing abnormal on the regular examination. The muscle test revealed in one of these a decided weakness of the gluteal muscles of one leg, showing that the case was classed as abortive only because the regular examination which was made was not delicate enough. In the other case, no departure was shown from the normal scale for a child of that age. This must therefore pass as a purely abortive case.

The method has probably a diagnostic value in detecting in cases which have been regarded as possibly abortive a decided local muscular weakness too slight to be detected by the ordinary manual examination.

Effects of Treatment. Muscle training as contrasted with simple massage showed that after a period of a few weeks, that is, when the treatments have been given for a sufficient time to demonstrate their effect, muscle training is superior to massage. This became evident after the third series of tests, and not after the second series, but a final conclusion cannot be presented on such short observation.

Muscle Training. As to the effects of treatment, in the time available for observation the following facts appeared: The chance of improvement in affected but not totally paralyzed muscles under expert treatment by muscle training was about 6:1, under supervised home exercises 3.5:1, under home exercises without supervision 2.8:1, while untreated affected muscles in these patients showed an im-

provement ratio of 1.9:1. These patients were nearly all affected in 1914, and represent cases at the end of the first year. They are all from the Vermont group and were treated there.

Spontaneous Improvement. The ratio of 1.9:1 chance of improvement in untreated muscle groups is apparently partly spontaneous improvement, but this question is so very important in the matter of prognosis that it demands further analysis, because in most of these patients other muscles were being treated, and the figures to be quoted indicate that untreated muscles are favorably affected by the systematic exercise of other muscles. There were available in the Vermont series only seven patients on whom repeated muscle tests were made who were not receiving treatment because the prescription had not been followed by the parents.

In these patients, in thirty-seven affected muscle groups there was improvement in nineteen and none in eighteen, a ratio of 1:1 of purely spontaneous improvement. These were all from the 1914 epidemic. The conclusion from this small group is that the treatment of one set of muscles by muscle training has a stimulating effect on muscles not under treatment.

These facts may well give us courage to undertake a more vigorous treatment in late cases. Knowing that at the end of one year spontaneous improvement is by no means at an end, perhaps we may find that it extends much later than this when our observations have extended. These observations on the recuperative power in the affected structures and how long it lasts appear to be of much practical importance.

Recovery of Power in Total Paralysis. The possibility of improvement in muscles totally paralyzed at the end of one year is also of much therapeutic importance. The following facts have been observed: Of forty-four totally paralyzed

muscles at the end of one year, 48 per cent (twenty-one) developed demonstrable power after two months of treatment. Of forty-four totally paralyzed muscles not receiving treatment, 27 per cent (twelve) developed demonstrable power at the end of two months, which shows again that spontaneous improvement has by no means stopped at the end of a year.

An observation shows how great this improvement may be and how late it may be obtained. A patient affected in 1911 was under treatment for two months, beginning last July, by supervised home exercises. His unaffected muscles in this time gained 70 per cent, while his affected muscles gained this and 400 per cent more (470 per cent). These muscles were severely affected. This great gain at the end of four years by simple home exercises and the avoidance of fatigue is, it seems, most unexpected and encouraging.

Types of Therapeutic Exercise. The usefulness of the method in throwing light on the value of different treatments is illustrated by the following observation:

It was asserted by an English writer that the most helpful exercise in infantile paralysis was to pull against the fully contracted muscle rather than to make resistance *against* the contracting muscle, technically that an eccentric exercise was physiologically more sound than a so-called concentric one. One observation showed that this advocated type of exercise was apparently harmful. A young girl with a moderately affected quadriceps muscle was given exercise of this newer type for two weeks, and lost 12.5 per cent of power; the regular exercises were then given for four weeks with a development of 50 per cent of power. It was not felt that it was proper to repeat this observation on other patients, so that it must remain only as an unproved suggestion.

OBSERVATIONS IN PRIVATE PRACTICE

The private patients under observation from the practice of one of us, some of whom have been under observation since June, 1914, offer good material for study, as being under the most continuous observation. Instances of the practical applicability of the method in these cases are as follows:

Case 3.—A girl of 10 had been affected since 1906, and when she came for treatment in 1911 she showed a mild equinus deformity of the right foot. After various attempts to stretch this it was operated on in January, 1915, by tenotomy, but the child, although improved, still walked poorly. Eight months later, in October, 1915, a muscle test showed decided weakness of the thigh flexors and extensors and an increasing weakness in the dorsal flexors of the right foot, which were getting weaker as the plantar flexors increased in strength. In October, 1915, exercises addressed to these muscles were prescribed, although the child had previously been doing exercises. In six weeks the mother reported that the progress since October had been far greater than at any time in the affection, and that the child could now "skip" on one foot and used herself very differently than she had ever done before, and the lameness had become very much less. An arm weakness had also been identified by the muscle test, for which exercises had been given with much benefit, an instance in which greater precision of treatment had demonstrated its greater usefulness.

Case 4.—A man of 22 was referred in the fourth week after his onset. He had involvement of the right leg and arm, walked with a limp and could not raise his right arm. The left arm appeared to be slightly weakened. Examination by the usual method showed extensive weakness in the left arm, very little power in the right deltoid, and a general involvement of the right leg. His right arm was put in a sling, he was cautioned against much walking and the use of the arm, and weekly muscle tests were made, showing a general slow gain, but no therapeutic exercises were allowed at first. At a test, October 4, an increase of 50 per cent in the power of the right gastrocnemius muscle was observed, and on questioning it was found that he had been daily rising on his toes as a trial. This seemed to indicate that he was ready for therapeutic exercise, on which he then began with

success. The test, October 26, showed a loss of 25 per cent of power in the wrist and finger flexors of the right hand, and it was found on questioning that he had been writing too much. This was stopped, and on the following week a return of the former power was found in these muscles, an instance of the information afforded by the test in directing routine and defining treatment.

Case 5.—A girl aged 8½, affected in 1913 with extensive paralysis of both legs, was walking with splints and crutches, and her muscle power was on the increase under daily muscle training. Nov. 18, 1915, there was a general loss of power in the legs. On questioning it was found that relatives had been visiting the family, and the child had been doing much more than usual, an instance of the deleterious effect of local and general fatigue.

These private patients are all tested at regular intervals, varying upward from one week, and it has been found that on the whole all are gaining in power and that the failure to gain in a special group or in general can be explained. Muscles gain and lose rapidly, a week often showing marked changes. As a rule, the muscles receiving the most attention gain fastest, and treatment is constantly to be modified in response to changes in the muscle readings. The more precise treatment is more satisfactory to those in charge of the case, and we believe of greater benefit to the patient.

CONCLUSION

No one realizes better than ourselves how very incomplete this report is, but we are not justified in making it otherwise. There has not been a sufficient interval since our first observations to warrant us in making sweeping assertions as to the value of different treatments. We have under way, however, a series of observations on the effect of the different therapeutic measures and on the effect of braces, and plaster of Paris in operative cases. These will in due time be presented, and meantime we only desire to call attention to a method of observation which we believe to be useful and to certain general conclusions which we believe to be sound.

THE SPRING BALANCE MUSCLE TEST*

By ROBERT W. LOVETT, M.D., Boston

AND

ERNEST G. MARTIN, PH.D., Boston

A MEANS of estimating the quantitative strength of muscular groups has been devised and has proved of practical use. It was formulated for the purpose of giving not only a qualitative but a quantitative value to the examination.^{1, 2, 3} It deals with muscular groups rather than with individual muscles, and is likely to be of assistance, not only in the examination of the individual cases, but also in the study of the phenomena of the disease and possibly in diagnosis. Data obtained by its use have already been published.⁴

That such a quantitative test was necessary became evident at the outset of the work in Vermont, references to which have just been given.

The method is designed to test, under conditions of constant position and leverage, by a series of spring balance pulls, the power of the muscles which govern the movement of the limbs. The value of the test consists in the possibility of duplicating exactly the conditions of the first test at succeeding ones, so that a definite idea of gain or loss in muscular strength can be registered in pounds. It is applicable for all tests of power in normal muscles, for determining loss or gain in power at stated intervals, and for the determination of the degree of initial weakness in paralyzed muscles. It has been applied to infantile paralysis cases for one year in consecutive tests varying in frequency from ten days to three months. The result has been an accurate register of general gain and occasional loss in these cases under

*Reprinted from *The American Journal of Orthopedic Surgery*, Vol. xiv, No. 7, pp. 415-424, July, 1916.

treatment. The record has the advantage of representing concisely, in figures, the results of very detailed muscular examination and of presenting at later examinations the initial and intermediate conditions of the case.

The accuracy of the test depends upon the training of two persons, an operator and an assistant, to coordinate the pull of the muscle and the registration of the pull on the scales, and upon the maintenance with exactness of the positions and leverage relationships outlined individually below. Accurate spring balance scales (No. 5 in Fig. 1)* are used, of 4 sizes: 1 to 4 lbs., graded in ounces; 1 to 30 lbs., 1 to 50 lbs., and 1 to 100 lbs. The readings are taken to the half pound except on the ounce scale.

The operator in general controls and maintains the correct position of the subject, stimulates the subject to innervation, braces and guides the limb tested, and calls the moment of give in the muscle tested through watching the action of the muscle itself. The assistant makes the pull along lines accurately determined, beginning and stopping under the direction of the operator. The same command directs the muscular pull of the patient and the scale pull of the assistant. In all cases where the position of the assistant makes this possible, the scale reading is taken by him at the moment when the yielding in the muscle is called by the operator. Except under special circumstances, plantar flexion is the only reading which the operator is required to make.

Twenty-two readings are taken, for each of which the best position of the subject for the accurate reading of the scales and for constant leverage in limb action has been determined experimentally. The order in which muscles are tested is immaterial except under conditions of weakness, but it is best that the order be constant so that all tests may duplicate each other as completely as possible. The apparatus required is shown in the accompanying illustration and

*See illustration facing page 254.

referred to by the number on each piece, as they come into use in the description of the measurements themselves.

In the lower extremity the test records the following movements: plantar flexion, dorsiflexion, inversion, eversion, adduction, abduction, hip extension, hip flexion, knee extension, knee flexion. The position of the operator and assistant in each movement is determined by their own convenience for fulfilling the other conditions of the test. The operations of these two individuals and the position and action required of the subject, for the measurement of these movements, are as follows:

Plantar Flexion. The subject lies on his back on a smooth table. The foot is braced against a three-to-one lever (No. 1). The scale hook is inserted in the ring of the lever upright. The lever must be adjusted so that the ball of the foot in maximum plantar flexion rests squarely upon the lever pad (No. 2), with the upright at an angle of from 60 to 80 degrees to the table. The lever is held in position by C-clamps (No. 3). The pull is made by the assistant from the head of the table with the scale horizontal and in line with the leg being tested, and is increased in intensity to the point where the muscular resistance is overcome. To prevent slipping on the table, the shoulders of the subject are held by the hip-braces (No. 4). The muscle gives at about 45 degrees of plantar flexion, with a rather sharp break in the resistance offered to the spring balance. All measurements of degrees are made to the plane of the table, unless otherwise specified. The operator guides the position of the foot, stimulates the patient to innervation, and calls the moment of break in the muscle to the assistant, for reading, or reads the scale himself. The reading of the scale must be simultaneous with this break.

Dorsal Flexion. The general position of the subject is the same. The foot should be flush with the end of the table, to give freedom of action to the assistant in making the pull,

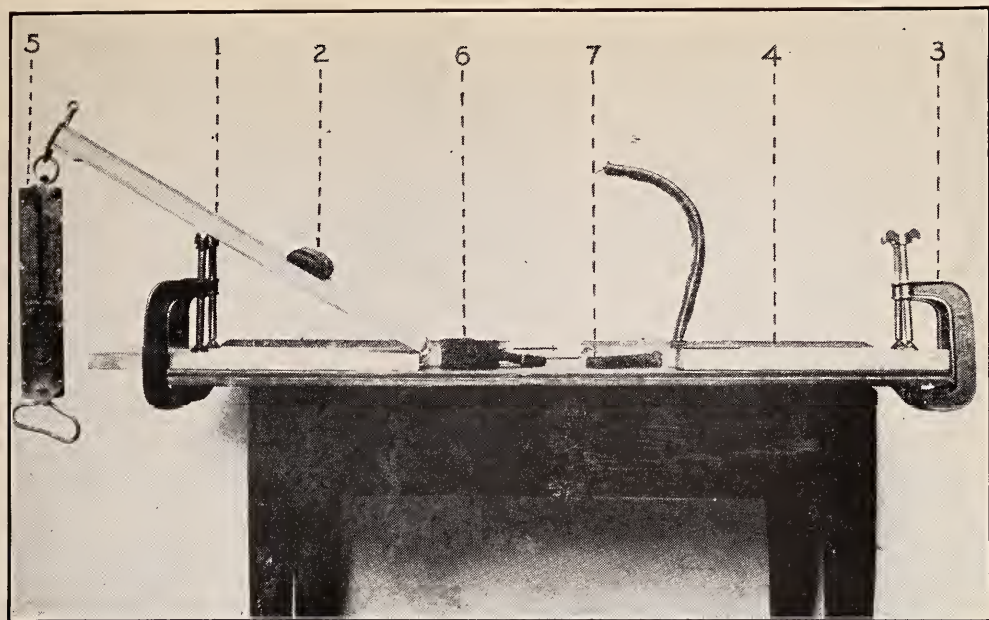


Fig. 1. Apparatus used in muscle test. The numbers in the illustration are referred to in the text.



Fig. 2. Test for dorsal flexion of foot.

and should be slightly lifted and braced by the hands of the operator, which encircle the ankle. The leather loop (No. 6) is placed across the toes at their metatarsophalangeal joints. With the foot in maximum dorsiflexion, the assistant makes the pull at right angles to the plantar surface of the foot, lowering the scale to maintain this angle as the foot gives. The muscle is watched by the operator, and the moment when it gives, called to the assistant, who takes the scale reading. (Fig. 2.)

Inversion. Body position of the subject the same as in dorsal flexion. The loop is across the inner surface of the great toe joint. The ankle is braced by the hands of the operator. With the foot at right angles to the leg, the foot is inverted and adducted as far as possible without inward rotation of the leg. The pull is opposite in direction to the muscular contraction, horizontal, and in the same vertical plane as the foot. The assistant swings the scale so as to maintain this relationship as the foot gives. The reading is taken by the assistant at the moment when the operator calls the break in resistance of the foot.

Eversion. General position as in dorsal flexion. Loop at the outer surface of the distal end of the fifth metatarsal. The foot is at right angles to the leg and is everted and abducted as far as possible without outward rotation of the leg. The pull is horizontal and in the same vertical plane as the foot, with a scale swing to maintain this position. The break is called and read as above.

Adduction of the Leg. No change in general body position of the subject. Two hip-braces (No. 4) are placed in line with the crest of the ilium on each side of the pelvis and attached to the table with the C-clamps. With one hand in the popliteal space and the other below the heel, the operator gently supports the weight of the leg, raises the leg about fifteen degrees from the table, and maintains the foot vertical to prevent leg rotation. The subject contracts the

inner muscles of the thigh so as to swing the leg inward across the median line about fifteen or twenty degrees. This angle of contraction is a matter of comfort to the subject and varies with the individual. The angle of elevation of the leg from the table must be constant. The subject, during the pull, braces the trunk with the hands by pushing against the clamps of the hip-brace on the side opposite to that being tested. The loop is placed just above the internal malleolus. The pull is outward, horizontal, and at right angles to the leg. It must swing so as to preserve this angle as the leg gives. The operator calls for the reading as the leg becomes exactly parallel to the median plane of the body. This reading can be taken in the same fashion with the loop at the knee just above the patella. The power here, with allowance for minor individual variations in leg length, doubles the ankle pull through halving the distance of the measuring spring balance from the fulcrum. The knee pull is used where the quadriceps is weak and it is difficult for the subject to maintain knee extension while making the adductor pull from the hip.

Abduction of the Leg. The details of position and bracing in this pull differ only in the direction of the muscular action, which reverses the bracing and the positions of the operator and assistant. The loop is just above the external malleolus. The subject contracts the muscles which abduct the hip so as to swing the leg outward from the median plane at an angle of thirty or forty degrees, according to individual comfort. The pull is inward, horizontal and at right angles to the leg. This angle and the angle of fifteen degrees of leg elevation must be maintained constant throughout the pull, exactly as in adduction. The operator calls for the scale reading as the leg becomes parallel to the median plane. The test is made at the knee where the quadriceps is weak.

Hip Extension. The subject lies on the side opposite to that to be tested, with the hips directly one above the other. The abdomen is braced against the hip-clamp used in abduction and adduction. At the lower end of the table, two C-clamps, across which a small board is placed for comfort, are used by the subject as a brace. He pushes against this with the foot of the leg not being tested, to secure steadiness. The trunk is braced forward, by the subject, by holding to the edge of the table with the hands. The operator maintains the position of the abdomen against the hip brace with one hand, and with the other supports the weight of the leg to be tested, and keeps the leg parallel to the table. The loop is at the knee across the popliteal space. The leg is placed in maximum extension with the knee straight. The direction of pull of the balance is slightly less than 90 degrees to the leg, being deflected towards the trunk, and is exerted horizontally. The angle of the pull must be constant throughout the movement. The operator calls for the reading as the leg crosses the line of the trunk, or if the muscle gives before this, the reading is taken when the muscle yields.

Hip Flexion. Side position and foot brace, as for hip extension. The small of the back is against the hip-brace. The subject maintains the rigidity of the trunk by pushing with the hands against the opposite hip-brace. The operator supports the leg parallel to the table, with one hand at the knee and the other at the ankle. The loop is at the knee just above the patella. The knee is well bent, and the thigh is flexed above the right angle. The pull is horizontal and as near as possible at right angles to the femur. The reading is taken when the muscle gives.

Knee Extension. The subject lies on the face on the table with the lower leg flexed at the knee and vertical to the table. The loop is at the ankle just proximal to the malleoli. The assistant stands at the head of the subject, bracing the

shoulder with one hand. The pull is horizontal, and parallel to the median plane. The operator braces the knee on the table with one hand, and with the other at the ankle limits the extension. The movement begins from the perpendicular position, and the effort of the subject to extend the leg and the pull of the assistant must start simultaneously at the command of the operator. Both pulls should begin slowly, and it is essential that the muscle pull and the pull of the spring balance should develop together in this test. The leg is not permitted to extend from the perpendicular position further than to within 75 degrees of the table. Greater extension than this changes the leverage and produces inaccuracy. The pull of the assistant continues until the knee is drawn back to the original position, the operator calling for the scale reading exactly as the leg crosses the perpendicular line. The quadriceps test is the most accurate of all tests as to repetition, but also the most liable to error if over-extension is permitted, before the balance pull begins to draw the leg back to the vertical position.

Knee Flexion. General body position of the subject and brace by the operator the same. Ankle loop reversed in direction and the assistant at the foot of the table. The subject places the leg in maximum flexion. The pull is horizontal and rotation of the hip should be minimized. The operator calls for the scale reading as the lower leg crosses the perpendicular position. If the reading is taken with the leg more than fifteen degrees beyond the perpendicular, accuracy is destroyed through change of leverage.

This group of leg tests can be made in half an hour where the subject responds easily to directions and the operator and assistant are accustomed to coordinate work. Every reading is repeated as a check. The readings agree very closely unless there is an error in technique. In the first test the pull generally rises slightly on the repetition because the

subject understands the requirements of the movement better the second time it is made.

In the upper extremity the test records the following movements: Pectoralis, latissimus dorsi, anterior deltoid, posterior deltoid, forearm extension, forearm flexion, wrist extension, wrist flexion, finger extension, finger flexion, thumb adduction, thumb abduction. In all the movements, the break is called by the operator and the scale read by the assistant. For the first four movements the loop is at the elbow just above the condyles of the humerus.

Pectoralis. The subject stands or sits, with the shoulders and the hips in the same vertical plane. If standing, he braces the thigh well against the table to prevent loss of balance. The arm is drawn as far as possible across the front of the body, just clearing the trunk, with the forearm in pronation. Any brace of the body with the opposite arm is permissible which does not disturb the plane of the shoulders and hips. The pull is horizontal and outward posteriorly at an angle of 30 degrees to the lateral plane of the body.

Latissimus Dorsi. The subject stands or sits, as above. the fist is closed, and with the dorsum of the hand towards the back, the arm is drawn as far as possible, across, behind the body, just clearing the trunk. The pull is horizontal and outward anteriorly at an angle of 30 degrees to the lateral plane.

Anterior Deltoid. Positions of the subject the same. The opposite hand holds to any support which does not elevate the shoulders. The arm being tested is raised to the level of the shoulder, and brought forward to an angle 30 degrees from the lateral plane of the trunk. The pull is backward and downward, establishing an angle of 60 degrees with the upper arm, and maintains this angle as the arm gives.

Posterior Deltoids. The subject stands or sits, as in the other shoulder tests. The arm is raised to the level of the shoulder posteriorly at an angle of 30 degrees to the lateral

plane of the trunk. The pull is forward and downward, establishing an angle of 60 degrees with the upper arm, and maintains this angle as the arm gives.

Forearm Extension. The subject lies on the back, with the arm at the side, and the forearm perpendicular to the table, against which the elbow rests. The hand is closed with the thumb pointing to the shoulder. The loop is at the wrist just proximal to the styloid process of the ulna. The assistant stands at the head of the table and braces with one hand the shoulder of the side to be tested. The operator braces the elbow on the table with one hand, and with the other at the wrist limits the extension of the forearm. The pull is horizontal. At the direction of the operator, the extension of the forearm and the pull of the assistant start together slowly. Extension is permitted to from 5 to 15 degrees from the perpendicular, and is overcome by the assistant. The call for the reading of the scale is made just as the forearm crosses the vertical line.

Forearm Flexion. No change in the position of the subject nor the bracing of the operator. Loop just proximal to the styloid process of the radius. The forearm is placed in maximum flexion with the elbow on the table, the hand closed, and the thumb pointing toward the shoulder. When the muscular power requires it, the foot brace described in hip extension is used in the same fashion by the subject to prevent slipping during the movement. The pull is horizontal. The operator calls for the scale reading as the forearm crosses the perpendicular line.

Wrist Extension. The subject extends the entire arm laterally and anteriorly, according to individual comfort. With the palmar surface of the hand vertical and the fingers extended, the wrist is put in maximum extension. The operator encircles the wrist with his hands, bracing the subject's arm in the extended position. The small loop (No. 7) is across the dorsum of the hand, just distal to the meta-

carpals. The pull is exerted horizontally and at an angle slightly less than 90 degrees to the hand, being deflected towards the wrist. The angle of pull must be constant, and to secure this the assistant swings the scale through an arc as the hand gives. The accuracy of the reading depends absolutely upon maintaining the direction of the pull and upon the correct placing of the loop, and is most important in this and the three following tests.

Wrist Flexion. With the arm well away from the side, the subject flexes the elbow according to comfort. With the fingers flexed at right angles to the palm, and the palmar surface of the hand in the vertical plane, the subject puts the wrist in maximum flexion. The small loop is across the palm at the crease formed by the finger flexion. The operator braces the wrist and arm in this position, encircling the wrist with both hands. The pull is horizontal and at an angle slightly less than 90 degrees to the dorsal surface of the hand. The angle of the pull must be maintained by an arc swing of the scales.

Finger Extension. The subject extends the arm as for wrist extension. The hip-brace is attached lengthwise to the side of the table. The palm of the hand well below the palmar crease is braced by the operator against the curved upright of the brace. The small loop is across the fingers dorsally, just proximal to the first interphalangeal joint. The pull is horizontal, and at an angle slightly less than 90 degrees to the extended fingers and deflects towards the wrist.

Finger Flexion. The position of the subject and the brace by the operator are the same. The small loop is placed across the fingers on the palmar surface, just proximal to the first interphalangeal joint. The palmar surface of the hand is vertical against the brace. The pull is horizontal and slightly less than 90 degrees to the proximal phalanges. The deflection is towards the dorsum of the hand.

Thumb Adduction. With the palmar surface down and the hand horizontal, the operator braces the extended fingers with one hand and the wrist with the other. The small loop is placed at the interphalangeal joint of the thumb. The subject adducts the thumb as far as possible under the palm. The pull is horizontal and at right angles to the thumb joint. The call for the reading is made by the operator just as the thumb appears from under the hand.

Thumb Abduction. General position of the hand and brace by the operator the same as for the preceding test. The subject abducts the thumb in the same horizontal plane as the hand. The position of the small loop is identical with that of adduction, but reversed in direction. The pull deflects downward from the horizontal just enough to escape the palmar surface of the hand. It is exerted at right angles to the thumb.

The complete arm test requires half an hour, and each reading is repeated as a check.

REFERENCES

¹MARTIN, E. G., AND LOVETT, R. W.: A Method of Testing Muscular Strength in Infantile Paralysis. Jour. A. M. A., October 30, 1915.

²LOVETT, R. W.: The Treatment of Infantile Paralysis. The Newer Aspects of the Problem, with Certain Conclusions Drawn from the Vermont Epidemic. Jour. A. M. A., June 26, 1915.

³LOVETT, R. W., AND MARTIN, E. G.: Infantile Paralysis in Vermont. A Report of the Progress of Cases Between January, 1915, and July, 1915. Vermont State Medical Journal, February, 1916.

⁴LOVETT, R. W., AND MARTIN, E. G.: Certain Aspects of Infantile Paralysis, with a Description of a Method of Muscle Testing. Jour. A. M. A., March 4, 1916.

A PLAN OF TREATMENT IN INFANTILE PARALYSIS*

ROBERT W. LOVETT, M.D.

THE successful treatment of infantile paralysis requires that the surgeon should have in his mind a definite plan covering all the phases of the disease, a plan based on the pathology in its various phases. We have at the outset a virulent acute affection with a high mortality, then comes a period of two years, during which we try to restore to their highest efficiency the affected muscles, and finally we meet in the later and so-called stationary stage of the affection the question of correcting deformity and restoring or improving function by operative measures. Few affections offer a wider range of requirements from a therapeutic point of view, and if when we are treating the first stage we bear in mind what may happen to the patient in the third stage, we shall treat the early stages better.

The muscle test spoken of in the paper is a means of quantitatively estimating the strength of muscles by means of their pull against a spring balance, and is not only useful in locating the existence of weakness in different muscular groups, but offers a means of estimating the gain or loss in muscular strength under given conditions.¹

The stages of the disease are as follows:

1. The stage of onset begins with the acute attack, and may be assumed to end when the tenderness has disappeared, a duration in general of from four weeks to three months. A hemorrhagic myelitis is present, widespread, and affecting chiefly the centers of motion. The centers of

*Read before the Section on Orthopedic Surgery at the Sixty-Seventh Annual Session of the American Medical Association, Detroit, June, 1916. Copyright 1916, Am. Med. Assn. and reprinted by permission from the Jour. of the A. M. A., Aug. 5, 1916, Vol. LXVII.

1. Martin, E. G., and Lovett, R. W.: A Method of Testing Muscular Strength in Infantile Paralysis, *The Journal A. M. A.*, Oct. 30, 1915, p. 1512.

sensation are also involved, the posterior root ganglia in experimental pathology showing the first changes, and the existence of tenderness must be accepted as evidence of an active process in the spinal cord. During this tenderness it is not physiologically reasonable to excite the peripheral connections of the affected nerve centers by massage and electricity. The former at times causes great increase of pain and soreness, and has nothing to recommend it at this stage, and there is no evidence whatever to show that electricity is of any value at this stage. Rest is the physiologic requirement, and the method of treatment that in practice works best, and the growing tendency to omit meddlesome therapeutic measures at this stage is hopeful. There is evidence that the use of hexamethylenamin in monkeys diminishes the risk of infection, but has no effect after the paralysis has occurred, and as the drug in moderation is harmless, it is extensively used in this stage. There is no serum or drug or proceeding that is known to abort the affection or limit the paralysis, although Netter of Paris has administered intraspinal injections of the blood of recovered persons with, he believes, benefit, but only in a small series of cases, and the proceeding is as yet wholly in the experimental stage.

During this stage the patient should be kept quiet. Joints will not stiffen, hopeless muscular atrophy will not occur, and by this proceeding the damaged cord will have the best chance to repair, and repair to the highest degree is desirable.

Deformities should be prevented by keeping the feet at right angles to the legs to avoid the most common deformity, a dropped foot. Toward the end of this period immersion in a warm salt bath is desirable and permits a degree of exercise to the affected limbs. Scoliosis begins frequently in this stage, and is often overlooked.

2. The second stage, or phase of convalescence, may be assumed to begin with the disappearance of the tenderness and to last for two years or more, at the end of which period the disease has become more or less stationary. The pathologic condition at this stage formulates the treatment. The hemorrhagic myelitis is subsiding, the perivascular infiltration which has blocked some of the spinal arteries is being absorbed, and these cells are resuming their function little by little, inflammatory products are being absorbed, and the clinical manifestation of these processes is expressed in what we all recognize as "spontaneous improvement," which begins when the tenderness disappears and lasts almost indefinitely, diminishing in its rate as the months pass.²

The clinical manifestation of the pathologic process is a motor impairment of muscles, widespread and in general erratic, more often a weakening than a complete paralysis. In the Vermont series of cases,³ manual examination of muscles showed the proportion between partial and total paralysis to be as $2\frac{1}{2}:1$, and the more delicate muscle test,² which detects slighter grades of weakening, found the proportion to be as 9:1.

Our problem at this stage therefore is to restore the maximum function to affected muscles, and to study carefully the measures most likely to accomplish this end. This point of view becomes especially important when we realize that muscular weakening is much more common than complete loss of power. It is a very important matter to the patient with a gastrocnemius muscle with only 20 per cent of the normal power whether that muscle ultimately regains 40 per cent or 90 per cent of its proper strength.

It becomes necessary then to consider those measures which are likely to prove most useful in bringing about the

2. Lovett, R. W., and Martin, E. G.: Certain Aspects of Infantile Paralysis, *The Journal A. M. A.*, March 4, 1916, p. 729

3 Lovett, R. W.: The Treatment of Infantile Paralysis, *The Journal A. M. A.*, June 26, 1915, p. 2118.

maximum improvement, and to comment on conditions likely to prove detrimental.

AMBULATORY TREATMENT

When the acute stage is over it is on the whole desirable to get the patient on his feet, that is, to institute ambulatory treatment. Prolonged recumbency is for a child unnatural and undesirable physiologically and mentally. The sitting position not varied by the upright position is of all most likely to lead to flexion deformity of the hips and knees and to dropped feet. The upright position induced by ambulatory measures is desirable not only because it antagonizes the conditions mentioned, but because the effort to balance on the feet instinctively excites to effort a large number of muscles not otherwise to be reached, and is a valuable form of "muscle training," a therapeutic measure to be mentioned later. On the other hand, this method is open to the objection of possibly fatiguing convalescent muscles and some authors advocate prolonged recumbency. In my opinion, the ambulatory method with proper avoidance of fatigue is on the whole the best to be pursued at the end of two or three months.

If the patient can walk without braces, so much the better. If apparatus is needed to permit ambulatory treatment it should be used, but worn only in walking and in early cases not continuously. The most commonly required form of apparatus is the Thomas caliper splint, which serves to keep the knees straight. Crutches may or may not be required. A good general rule with regard to apparatus is that it should be used when the patient cannot stand without it, or if in standing or walking a position of deformity is assumed, because deformity leads to stretching of soft parts, and if persisted in to permanent bony changes.

The two conditions most frequently overlooked which lead to serious results are weakening or paralysis of the

abdominal muscles and scoliosis. In the writer's opinion, when these occur the use of a cloth corset or plaster jacket is imperative from the time that the first stage is over.

A patient who has been long in bed when first put on his feet is often unable to balance even if he has sufficient muscular strength, and the problem of cultivating equilibrium in these cases must be taken up by itself and patiently persisted in.

Having thus formulated the matter of ambulatory treatment, the question next arises as to those therapeutic measures which are likely directly to have a favorable effect on the muscles. These are (1) massage, (2) electricity, (3) heat, and (4) muscle training.

1. Massage is of value because it empties the veins and lymphatics and thus promotes the flow of blood to the limb, and because it apparently retards muscular atrophy and promotes muscular tone. More than this, however, is not to be expected of it. It does not promote the passage of nervous impulses from brain to muscle, and its action must be considered purely local. Given roughly or for too long a time it is detrimental and retards progress, and its overuse is probably responsible for much harm.

2. Electricity has been much discussed, and in the absence of definite data one must fall back on personal experience. Faradism causes a mild muscular contraction, and may be a useful form of gentle exercise. It is disagreeable, and to young children often a source of terror. The galvanic and newer forms of currents are assumed to have a beneficial effect in general, but in many years' experience in treatment with and without electricity (used often on one side of the patient only with the other side as a control) the writer has never been able to satisfy himself in a single case that it was of any value. Certain recent experimental

work on the retardation of atrophy in denervated muscles is of interest:⁴

It is practically certain that if electrical stimulation has a beneficial effect, the optimal effect will be with that current which is strong enough just to cause contraction. In the ordinary methods of stimulating muscles through the skin, whether by unipolar or bipolar methods, with currents of long or short duration, the intensity of the current is much greater in the superficial than in the deep fibers, and we think it doubtful whether the latter can be stimulated without using currents injurious to the former.

That the use of electricity has done much harm is undoubted, because not only is the use of strong currents admittedly injurious, but the routine use of electricity often deludes the physician and parents into thinking that the child is receiving adequate treatment while measures of admitted value are neglected.

3. Heat is of value either as radiant heat from electric bulbs or by some form of oven, because it raises the temperature of the limb, and thus offers more favorable conditions for muscular contraction, and because it stimulates the flow of blood to the limb.

4. Muscle training is in the writer's opinion the measure of the greatest value at this stage, and this stage is important because, however operative the surgeon may be, he will during these two years use nonoperative treatment.

Muscle training attempts to drive an impulse from brain to muscle to enable it if possible to open up new paths around affected centers in the cord. The connection between these centers with each other and between the centers and the muscles is most extensive and complex,⁵ and the facts given as to the predominance of partial paralysis show that as a rule the entire nervous control of a given muscle is not wiped out as a whole, but only in part. On this basis

4. Langley and Kato: *Jour. Physiol.*, 1915, xlix, 432.

5. Bing: *Compendium of Regional Diagnosis in Affections of the Brain and Spinal Cord*, New York, 1909.

rests the claim of muscle training, a measure which in the opinion of the writer is one of the most powerful factors in determining ultimate muscular function.

As to the efficacy of this treatment, the following data were observed in Vermont by means of the muscle test. The period covered was three months:

The chance of improvement in affected but not totally paralyzed muscles under expert treatment by muscle training was about 6 to 1, under supervised home exercises 3.5 to 1, under home exercises without supervision 2.8 to 1, while untreated affected muscles in these patients showed an improvement ratio of 1.9 to 1. These figures represent cases at the end of the first year. They are all from the Vermont group and were treated there.

In order to determine just what might be expected from the treatment by muscle training an analysis was made of the progress of all patients in my private practice during the past winter who had had two successive muscle tests at times widely enough separated to warrant any conclusions as to their progress. No cases were omitted, and the patients, as indicated in the accompanying table, are divided into two classes, those coming daily to the office for treatment by an expert assistant, and those whose treatment by muscle training was prescribed at the office but carried out at home by some unskilled person.

One difficulty presented itself in the analysis. Certain cases were regarded as having no power at all at the first examination, and in these cases for purposes of computing it was assumed that the children had power of one-eighth pound, not sufficient to move the scale. It was found that a child with one-fourth pound of muscle power would, however, move the scale, and these were the ones that were noted at the initial observation as having a trace of power. The percentage gain of each muscle was then reckoned, and the figure given in the table represents the average percentage of gain of all the muscles. If the paralysis was uni-

INFANTILE PARALYSIS IN VERMONT

RESULT OF MUSCLE TRAINING

<i>Age</i>	<i>Time Since Attack</i>	<i>Interval Covered by Tests</i>	<i>Average Total Gain of Affected Muscles, per Cent.</i>	<i>Average Monthly Gain of Affected Muscles, per Cent.</i>	<i>Apparatus Worn</i>	<i>Region Recorded</i>
------------	----------------------------------	--	--	--	---------------------------	----------------------------

Patients treated daily at office by skilled assistants

22	1 mo.	6 mos.	197	24	Sling	Arms
8	3 mos.	5 mos.	82	16	Corset, crutches, braces	Legs
9	3 mos.	6 mos.	146	21	None	Legs
9	21 mos.	10 mos.	200	20	Plaster jacket, crutches, braces,	Legs
14	4 mos.	4 mos.	688	172	Corset, braces, crutches	Legs
8	15 mos.	7 mos.	702	100	Corset, braces, crutches	Legs
8	3 mos.	6 mos.	184	30	Corset	Legs

Patients treated at home by relatives or nurses (unskilled)

10	1 yr.	6 mos.	13	2	High heels	1 leg
30	5 mos.	1 mo.	44	44	Sling	1 arm
24	6 yrs.	3 mos.	12	4	None	1 leg
10	2 $\frac{1}{4}$ yrs.	8 mos.	108	13.5	Corset, plate . . .	1 leg
11	6 yrs.	5 mos.	89	17	Plaster jacket, braces	Legs
4	1 yr.	7 mos.	30	4	Brace	1 leg
11	9 yrs.	2 mos.	33	17	None	Arm and leg
10	3 yrs.	4 mos.	16	3.5	High heel	1 leg
10	5 yrs.	7 mos.	None	None	High heel	1 leg
14	8 yrs.	8 mos.	67	8	Plaster jacket, brace	1 leg
16	2 mos.	3 mos.	620	206	Brace and crutches	1 leg
8	1 yr.	7 mos.	202	28	Braces and corset	2 legs, 2 arms

lateral, the per cent of gain in the unaffected limb was deducted from the gain of the affected side and only the excess counted.

Deformity occurs in many forms, but the therapeutics of it are easily formulated. In the earlier stages it is generally to be avoided by preventing persistent malposition. If fixed deformity exists it must be removed before undertaking treatment, nonoperative or operative. The neglect of this rule is one of the most frequent causes of failure of treatment. Deformity is corrected by stretching by hand, by plaster or by apparatus, by forcible stretching under anesthesia, by tenotomy, fasciotomy, myotomy or osteotomy, the mildest measure that will suffice being the soundest and best.

Stretched muscles are at a great disadvantage so far as recovery goes, as pointed out years ago by Charles Fayette Taylor, and later by Robert Jones. The best example of this is in gastrocnemius paralysis, most efficiently treated when it exists alone by very high heels, throwing the muscle out of use and preventing stretching.

Fatigue and overtreatment by massage and exercises are detrimental factors of the highest importance too little attended to. This has been especially brought out by the studies by means of the muscle test, which have shown that a surprisingly small amount of exercise was detrimental to convalescent muscles, and in some muscles returning power has been wholly abolished by overuse. The advice often given to use affected limbs as much as possible is in the opinion of the writer the worst advice that can be given. It is difficult to underuse such muscles, but fatally easy to injure them by overuse.

3. The third stage is generally called the stationary stage, and begins about two years after the onset. The requirements of the preceding stage as to the care of the muscles, etc., still exist, but are less urgent. In cases which have not

been properly treated earlier, muscle training may accomplish much, even in cases of long duration. The requirements as to apparatus remain much the same throughout.

The dominant requirements of this stage are operative, and are first the correction of deformity, a matter already discussed, and second, operations to improve function and secure stability.

Operations to improve function are by all experienced surgeons deferred until at least two years after the onset (and by some men several years) in order to permit recovery of muscular power to become as great as possible and to enable the mechanical conditions in the affected limb to become clearly defined before operating.

Tendon transplantation⁶ is the most brilliant of these measures. It implies the existence of one comparatively normal muscle in the region to be operated on. Simple operations have replaced complicated ones, periosteal insertion is used, silk extensions are in common use, tendons are passed in the subcutaneous tissue, and prolonged after-treatment is the rule, unrestricted use not being allowed under one year from operation.

Nerve transplantation,⁷ which is the other operation to improve function, has not been generally used because the most skillful operators in this field have not reported a large proportion of satisfactory results, and also for the reason that the operation is advised at so early a period in the disease.

OPERATIONS TO IMPROVE FUNCTION

Arthrodesis⁸ has lost favor because of the entrance in the field of operations yielding better functional results, and

6. Lange: München. med. Wehnschr., 1902, No. 1; Ztschr. f. orthop. Chir., xxix; Ztschr. f. ärztl. Fortbild., 1905, 22. Vulpius: Deutsch. med. Wehnschr., 1912, xxxvi. Lovett: Boston Med. and Surg. Jour., 1910.

7. Spitzzy: Handbuch der Kinderheilkunde, Lange and Spitzzy, Leipzig, 1910, p. 310; Ztschr. f. orthop. Chir., xiii. Osgood (review): Boston Med. and Surg. Jour., June 30, 1910. Vernicchi: Arch. di ortop., 1910, xxvii, 337. Kilvington: Brit. Med. Journ., April, 1907. Deroux: Lyon chir., December, 1912.

8. Jones, Robert: Tr. Int. Cong. Surg., 1909, xvi.

the most experienced surgeons do not favor it in the ankle until after late childhood, if at all. At the knee it is always questionable and dangerous in early childhood. In the hip it is desirable, but often unsuccessful.

Astragalectomy,⁹ on the other hand, has gained in favor, and although originally introduced by Whitman only for talipes calcaneus, it is now widely used where arthrodesis would formerly have been performed. A transverse section of the foot devised by Davis¹⁰ deserves mention as being useful in calcaneus deformity.

Silk ligaments¹¹ are used because silk left in the tissues becomes coated with fibrous tissue and serves as a ligament. This is especially used to correct the dropped foot by passing several strands of silk anteriorly from the tibia to the tarsus. It is a brilliant operation when successful, but has often failed, probably because too little silk has been used. In a child of 8 or 10 one should use six to eight strands of No. 12 silk.

Tendon fixation¹² or tenodesis has lately been revived by Gallie, and is extensively used. The paralyzed tendons are sewed into grooves in the bone, thus being transformed into ligaments, to correct deformity and check excess of motion. Stretching may occur, but the operation seems to have a field of usefulness.

Two of these operations are frequently combined, as, for example, tendon transference and silk ligaments, and similar instances.

SUMMARY

This paper is a plea for a definite uniform plan for the treatment of infantile paralysis in all of its stages, for a direct attack on the disease based on its pathology, and for persistency and precision in that therapeutic attack, with

9. Whitman, R.: *Ann. Surgery*, February, 1908; *Am Jour. Med. Sc.*, November, 1901.

10. Davis, G. G.: *Am. Jour. Orthop. Surg.*, October, 1913, p. 240.

11. Lange: *München. med. Wchnschr.*, 1906, li; *Ztschr. f. orthop. Chir.*, xvii, 266.

12. Gallie: *Am. Jour. Orthop. Surg.*, January, 1916.

special care as to the avoidance of fatigue from overexercise or overtreatment. It is the belief of the author that nowhere in orthopedic surgery does the difference between the best and indifferent treatment have more effect on the ultimate result than in this disease.

234 Marlborough Street.

HISTORY OF THE AFTER-CARE OF POLIOMYELITIS IN VERMONT

By ROBERT W. LOVETT, M.D.

THE history of the after-care of poliomyelitis in Vermont is of interest as it was the first state-wide work undertaken anywhere in this country and is today the most fully developed scheme for the after-care of poliomyelitis on a state-wide scale which exists.

In 1914 there occurred in the northern part of Vermont an epidemic of great severity, with a very high incidence across the northern half of the state, and from this epidemic 306 cases were reported. Undoubtedly not all the cases were reported, but of those that were the mortality rate was 23½ per cent, so that 226 cases at the close of this epidemic were left with varying degrees of paralysis. This was the condition that faced the State Board of Health at the close of the epidemic and it was obvious that, unless something was done in the way of treatment, the amount of resulting disability and disabling deformity would be very great.

Vermont is essentially an agricultural community with comparatively few inhabitants; large cities are few; and the specialties of medicine not highly developed except in one or two medical centers. The state consists of 9,565 square miles of mountainous country and many of the less traveled roads are difficult to negotiate, even in the best of weather.

Knowing the situation, an anonymous friend of the state placed at the disposal of the State Board of Health a generous sum to be used in the study of the prevention and care of poliomyelitis. It was decided to attempt to furnish care to as many of these children as possible, and in December, 1914, I was asked by the State Board of Health to go to Ver-

mont and undertake the work. On making a survey of the situation, it was found to be impossible at this season, or in fact at any other time, to see the cases individually in their homes, and it was decided to hold clinics at certain centers in the infected regions to which the cases might be brought for examination and direction as to treatment.

Poliomyelitis is a condition which is attended by high mortality in the acute disease. When the acute stage is over, spontaneous improvement immediately begins and continues for a time, after which disabling deformities are likely to set in. As is well known, in a certain number of instances these deformities cause such disability that the patient becomes a dependent or in any event fails to obtain the degree of recovery which it is right to expect would be possible under favorable conditions of treatment.

After considering the subject, the best solution seemed to be to examine the cases in consultation with the family doctor, who was invited to come to the clinic, and to advise him as to the measures to be followed and to instruct the family in carrying out such exercises and manipulations as seemed advisable.

The first series of free, public clinics for poliomyelitis in this country were conducted in this way in December, 1914, and January, 1915, in the state of Vermont. I was aided in this work by my assistant in private practice, Miss Wilhelmine G. Wright. The most important centers were visited, —Burlington, Montpelier, Barton, Rutland, and St. Albans, 5 in all. Two hundred and twelve cases were examined at this time and physicians and parents were advised as to the care and treatment to be given each patient. There was no provision made for follow-up work after the patient was seen at the clinic, a matter which later proved a serious defect in the work, but at the outset the whole proposition was so very uncertain that it seemed best to progress slowly and see what measures were possible.

During the following summer,—in July, 1915,—clinics were again held in the same places and we had the advantage of the presence of Dr. E. G. Martin, at that time Assistant Professor of Physiology in the Harvard Medical School, now Professor of Physiology in Leland Stanford University. Dr. Martin was interested with me in evolving a quantitative method of estimating muscle strength which we used at this time and which has been very extensively used since. Up to this time the strength of the muscles was estimated by hand and the condition was much as if one were studying typhoid without the advantage of a thermometer. The method proved to be practical and much of the information derived from the study of the Vermont cases was due to the more accurate method afforded by the so-called "spring balance" test.¹

Miss Wright and also Miss Janet B. Merrill of the Children's Hospital, Boston, came to assist at the clinics. One hundred and twenty-two examinations were made at this time. The patients who had followed the treatment prescribed at the previous clinic showed marked improvement, nevertheless it was realized that even better results could be obtained if the advice to parents could be supplemented by the supervision of a trained field worker who could visit the homes and follow up the treatment prescribed. Miss Merrill was the first to hold this position and spent two months in Vermont during the summer of 1915 after the clinics, making her headquarters in Burlington.

By December, 1915, enough new cases had been seen at the summer clinics or had applied for treatment to bring the number up to 243 for the year 1915. On this number of cases 334 clinical examinations were made either by the surgeon or his assistants.

The difficulty of getting about the country with the rather inadequate train service restricted the work to the care of

¹Described in detail on page 252.

those patients who lived in or near the larger centers. Miss Merrill, working in cooperation with the family physician, gave personal supervision to the muscle training of these patients and taught some member of each family to give the exercises, massage, etc., in her absence. The value of this work was demonstrated beyond question by the results of the muscle test given to each patient by Dr. Martin at the end of Miss Merrill's stay. These tests showed that the improvement in each case was in exact ratio to the amount of training and supervision given.

Incidental to the work, some cases which proved not to be poliomyelitis were brought for examination and have been included in these numbers.

Miss Merrill took up the field work again in the summer of 1916. This time the use of an automobile enabled her to cover a larger area and to save much valuable time. Assisted by Miss Merrill and Miss Miriam T. Sweeney, I held a series of clinics in July. For a few weeks after the clinics Miss Sweeney remained in Vermont to assist in the field work by taking charge of the muscle training of the patients in Burlington. No work was done by the After-Care Department during the autumn, but in the winter Miss Merrill returned to Vermont and held bi-weekly clinics in the larger centers. This made it possible for her to give careful supervision to such cases as could be brought to her, and to see each patient at least once in two months. During 1916, 42 patients who had not been examined previously came to the clinics and were taken under treatment.

In 1917 a severe epidemic occurred in the Montpelier district and, because of the additional demands, it was advisable to have two field workers. Miss Merrill was unable to continue with the work and resigned after starting Miss Helen King and Miss Rebecca Selfridge in the field. Miss Selfridge concentrated on the new cases in and about Montpelier while Miss King took charge of the work in the rest

of the state. Clinics were held by me in August with the assistance of the field workers, and 186 examinations were made. During this year 119 new patients were admitted to treatment, 79 of which were cases of recent onset.

Prior to 1917 the work was of necessity restricted to the larger centers and the territory immediately adjacent, and it was limited to those patients who had applied to the clinics for treatment. During 1917 the first effort was made to investigate the cases near each center which had not reported to the clinics for examination.

In 1918 the war depleted the staff to such an extent that the work of the department was practically brought to a standstill. Miss King resigned to take up reconstruction work in France and I was in the army, so that no clinics could be held. Only 14 new patients were admitted to treatment during the whole year. Miss Selfridge bore the entire burden of the work alone until the end of the summer when she resigned. From that time until the following spring of 1919, the department ceased functioning and the patients were without supervision.

Meanwhile, the Board of Health was besieged with requests for help,—existing deformities were increasing and new ones were developing; braces had been broken or outgrown; new cases needed examination and advice,—so that when the department resumed its activities again in May, 1919, in charge of Miss Bertha E. Weisbrod, the situation was acute. An accumulation of work in arrears required attention in addition to the need for meeting each day's new demands; a certain discouragement engendered in some of the patients by the interruption of the work and the uncertainty of its continuance must be met and overcome; 500 or more cases scattered over the northern part of the state must be followed up,—these were some of the conditions which faced the new régime. An immediate effort was made to get in touch with as many patients as possible and to per-

suade them to attend the clinics, with the gratifying result that 418 reported for examination during the year. This was more than had ever been examined in any one previous year, and more than double the number of examinations in any of the three preceding years. During 1914-1915, 334 clinical examinations had been made, but this was the year of the epidemic when excitement was rife and the parents seized every opportunity for help. In 1916-17-18, there were fewer patients at the clinics for two reasons: first, many had been discharged as cured; second, a natural reaction had set in,—the parents had grown used to the situation and were inclined to let things drift. The increased number in 1919 shows how fruitful was the effort to counteract this discouragement and stimulate interest.

The department inaugurated a new policy with regard to the follow-up work during 1919. Before this time visits to the homes of the patients had been restricted to the summer months and while clinics were held by the supervising nurse during the winter, only such patients as could be brought to them could be kept under constant supervision. In 1919, the department not only held winter clinics but also continued to make home visits during the entire year. This is still the policy of the department and the follow-up work in the homes is carried on without interruption even through the most severe Vermont winter.

In 1919 the first clinics since August, 1917, were held by me in July, and a sixth center, St. Johnsbury, was added at this time. Three additional clinics were conducted by Miss Weisbrod during the year, and the examinations showed conclusively that the previous work had been well worth doing. The patients who had followed the prescribed treatment of rest, massage, and carefully graded muscular exercises had made great gains. At this time there were comparatively few cases of recent onset, and a number of the old cases had reached the stage where operations were advis-

able and braces more essential than continued muscle training.

These two outstanding features offered new problems to be solved. Hitherto, braces and other apparatus had been made at the Appliance Shop of the Children's Hospital in Boston. This shop was overcrowded with work and often it was necessary to wait undesirably long before orders could be filled. Therefore, it was decided to try to make the appliances for the Vermont cases in that state. Mr. Frank A. Dresser, an instructor in the Engineering Department of the University of Vermont, an expert in mechanics, became interested and learned to make the various types of braces needed. The results of the experiment were so satisfactory that the appliance shop became a permanent institution and since it started the braces have been delivered more promptly and have fitted most satisfactorily. Mr. Dresser has made various improvements in the braces which have added to their efficiency and to the comfort of the patients. At the present time the department is trying one of the newer metals, duralumin, which is two-thirds lighter than steel and promises to be of great value for braces in this work, since the wearing qualities are proving satisfactory.

Meanwhile, a new problem had arisen in the matter of cases requiring operation. At the end of the second year and sometimes earlier, deformities begin to arise in poliomyelitis, such as drawing up of the knee or hip, or similar conditions which, although preventable, occur in a certain number of cases. To allow these deformities to go uncorrected is to bid for disability, and the relief of them in the majority of cases is a simple matter if operated on under the best conditions. Up to 1919 a few urgent cases had been operated on either in hospitals in Vermont or had been sent down to the Children's Hospital in Boston but the problem was becoming increasingly troublesome. In 1919 it became evident that some definite plan would have to be adopted. The mat-

ter presented certain difficulties, however, because many of the parents were opposed to having operations performed, and a great deal of time had to be spent in educational work before the necessary permission could be secured. When this was finally achieved, it was found that the hospital accommodations hitherto available were inadequate for the increased number of operative cases. Some of the patients had been operated on at local institutions by local physicians, several special cases were taken to the Hospital for the Ruptured and Crippled in New York to be under the care of Dr. Armitage Whitman, the majority of those under 12 years of age were operated on at the Children's Hospital in Boston by my colleague, Dr. F. R. Ober, or myself; but all of these hospitals had long waiting lists and additional accommodations were necessary. Moreover, in order to secure the best results with the patients from the rural districts it was important to keep them close at hand until the plaster casts had been removed and all apparatus had been fitted. The remoteness of their homes made it impossible to give them proper supervision in any other way. Therefore it was decided to secure hospital space in Vermont where Dr. Ober could operate and where the post-operative care could be continued as long as necessary.

As a result of this decision, in the winter of 1919 two wards of six beds each were rented from the Proctor Hospital in Proctor, Vermont. Miss Madeline Gibbs, a graduate of the Children's Hospital and especially trained in orthopedic work, was placed in charge and the department procured all the necessary orthopedic apparatus.

Twelve patients were taken to the hospital on December 5, and on December 7 Dr. Ober and his staff arrived from Boston. Dr. Ober operated for two days and stayed one more day to observe the condition of the patients. After that he returned for four week-ends to perform subsequent operations and to change plaster casts. By the middle of

March the last patients were discharged. The venture was successful in every respect and all the patients showed great improvement. The change in these children, together with the fact that they had been so well cared for and actually happy while in the hospital, did much to mitigate the dread of operations and to remove the prejudice against hospitals in the parts of the state from which these little patients had come. The highly satisfactory results in these operative cases were due in part to the fact that the patients were given the best of post-operative care in their homes after their discharge from the hospital. Each case was followed up at home by the field worker and kept under careful supervision until everything possible had been done to insure a complete recovery. This plan of providing proper post-operative care in the homes, initiated in 1919, has become a permanent part of the regular work of the department.

In 1919, 36 patients were operated on,—12 at the Proctor Hospital and the remaining cases either in hospitals in Vermont, the Hospital for the Ruptured and Crippled in New York, or the Children's Hospital in Boston.

Throughout 1919 Miss Weisbrod carried on the field work entirely alone, but in the autumn a secretary, Miss G. F. MacBride, was provided to assist her in the office and with the records. During the year, 270 cases were under supervision, 69 new patients applied for treatment, there were 418 examinations at clinics, 330 home visits were made, and 87 pieces of apparatus were fitted.

On May 1, 1920, an assistant field worker was added to the staff, the position being filled by Miss Marjorie Hickok. It will be remembered that prior to 1919, the field work had been restricted to the cases comparatively near the clinic centers and that in 1919 attention was directed to following up *all* the cases in and about those centers and getting them to report for examination. In 1920, a special

effort was made to locate *all* the cases on record in every part of the state, in addition to carrying on the regular work. It was found that 250 patients had not been seen for two years or more. All but 24 of these were traced. Even the most remote localities were visited and the condition of each patient, the treatment prescribed, care in following it, extent to which disability interfered with education and self-support, and the approximate amount of improvement, were noted on each record in order that a statistical study might be made, showing what the work had accomplished. The preliminary work was completed by September 1st, at which time Miss Catherine D. Jones, of the School of Public Health of Harvard University and Massachusetts Institute of Technology, came to Burlington to compile the figures. This study of the records was continued through 1921, and the results are presented in the statistics given at the end of this article.

During the summer of 1920, the usual clinics were held, but this time in charge of Dr. Ober, as I was in Europe, and a new clinic center was added at Barre. The first of November, the assistant field worker, Miss Hickok, was obliged to resign because of ill health and Miss Weisbrod carried on the work alone during the rest of that year. After the summer series of clinics, two subsidiary ones were held by Miss Weisbrod with the addition of still another center, Windsor, making 8 clinic centers in all. At the 1920 clinics not only were the usual examinations given and treatment prescribed but all apparatus was adjusted and plaster casts were made for jackets. Sixty-eight new patients applied for treatment during the year.

In view of the fact that the age limit for admission to the Children's Hospital in Boston was 12 years and because of the increasing number of patients in need of operation, it seemed advisable to secure accommodations in some other hospital where Dr. Ober might operate and where the de-

partment could be in complete charge of the post-operative care. Kimball Cottage, a hospital building connected with the New England Hospital for Women and Children, in Boston, was finally rented. The New England Hospital furnished heat, light, food, and laundry. In all other respects the organization was wholly independent.

This hospital was opened on November 13, 1920, with 36 patients from 3 to 26 years of age. These patients were all taken to Boston on the same day by railroad. The parents took the children to the nearest railroad station where they were met by workers who cared for them during the journey. The Volunteer Red Cross Motor Service carried them from the North Station to the hospital.

Forty-eight patients in all were operated on at Kimball Cottage. Of these, 35 were treated for deformities caused by poliomyelitis and 13 for deformities arising from other causes. Twenty-two had tonsils and adenoids removed by Dr. Calvin B. Faunce. Dr. George Derby examined the eyes of all the patients and fitted 6 with glasses. The teeth of all the patients were cleaned and all necessary filling and extracting was done by Dr. Johnson.

In addition to the excellent physical care given to the children, everything possible was done to keep them contented and happy. Children of school age were supplied with teachers so that all of them were able to keep up with their classes. Through the kindness of friends of Vermont in Boston, the patients were frequently taken on automobile rides or in the electric cars, which proved even more of a treat to some of these youngsters from the rural districts who had never even seen a street car before. Battleships, submarines, all these things were new to them, and convalescence instead of being just a number of dreary days to be dragged through became a thrilling adventure from beginning to end. Their escorts declared that it was more fun to take these keen, interested patients around the city than

anything else they could do, and the children themselves derived as much mental stimulus as physical benefit from their stay in Boston.

Each year from the beginning the work had presented its own problems and in time a satisfactory solution for each had been found. Gradually the scope of the work had broadened and the area covered had increased until in 1920 practically the whole of the state was under supervision, the surgical care was as good as we could make it, hospital and post-operative care had reached a high point of efficiency, but a serious problem still presented itself for solution.

The home surroundings of some of the patients were such that it was impossible to secure the best results if the children must be sent back from the hospital to face such living conditions. Sometimes it was merely a question of remoteness, the child could not be visited often enough to insure proper care; again, the financial status of the parents or the demands of a large family made it impossible for them to give the necessary treatment at home; and what was very important, in many cases a combination of these circumstances made it impossible for the children to attend school at all. During this year, one of the dreams of those interested in the after-care work was realized and provision was made for just these children. Through the generosity of a public spirited citizen of Vermont, Ormsbee House, a school and home for crippled children, was opened in Proctor, Vermont, on January 5, 1921. This school can take care of 18 children between the ages of 6 and 12, and although it cannot meet the entire need because its size does not enable children over 12 to be kept, it is ideal for those to whom it is available. The school is free to those who cannot afford to pay, so that the children who need it most may enjoy its privileges.

The house was built especially for these children. In addition to a staircase, an incline to the second floor is provided so that children with long braces and crutches may ascend easily. There is a well-equipped gymnasium where the exercises and muscle training are given. The large, sunny playroom has a sun-parlor at one end, an open fireplace, a piano, a victrola, couches, well-filled book shelves, and toys to delight the heart of any child. Just outside is a garden of their own and even an automobile is at hand for their use. Everywhere there are evidences of thought for the convenience and happiness of the pupils. Every bit of equipment is so carefully adjusted to their special needs as to render the children as independent of aid and as much like normal children as possible. A real home atmosphere pervades the school and there is nothing remotely suggestive of institutional life about it. A nurse, Miss Ina Allen, who is admirably adapted to the work, is in charge and under her careful supervision they are kept busy and happy from morning until night. Lessons are given to them in shorter periods than in the schools for normal children and in addition to the regular school work, cooking, sewing, and handicraft are taught, as well as music for those of special ability.

Of the children at Ormsbee House, five of the eighteen could only crawl when they originally came to the clinics and now they walk amazingly well even though braces and crutches are still necessary. All of the children show great improvement from month to month, physically, mentally, and morally, and they are happier in every way than in their own homes, for here they not only have the best of care and understanding but are spared the continual struggle to keep up with physically normal children which would be inevitable elsewhere.

During 1920, 68 new cases applied for treatment, 482 home visits were made, 162 pieces of apparatus were fitted, 131 braces were adjusted, orthopedic corrections were made

to shoes in 110 cases, exercises were taught to 144 patients, and 72* patients were operated on.

In the spring of 1921 the position of assistant field worker, which had been vacant since Miss Hickok's resignation in November, 1920, was filled by Miss Marguerite Belt of the Children's Hospital in Boston. In October Miss Belt was transferred to Kimball Cottage while Miss Margery C. Kerr became assistant field worker in her place.

Clinics were held in July with the addition of a ninth center at Proctor. I was in charge of the clinics at Rutland, Proctor, Burlington, and St. Albans, and Dr. Ober in charge at Barton, Montpelier, St. Johnsbury, Barre, and Windsor. Miss Weisbrod conducted subsidiary clinics in October and May.

In 1921, 463 patients were under supervision, 79 new cases applied for treatment, 568 home visits were made, 172 pieces of apparatus were applied, 114 pieces of apparatus were adjusted or repaired, 109 orthopedic corrections to shoes were made, and 71* cases were operated on.

The method of caring for the operative cases in 1920 had shown such excellent results that Kimball Cottage was again rented from the New England Hospital and was opened on December 1, 1921, with 34 patients. The Red Cross Motor Corps again helped to transfer the patients from station to hospital and the societies of the "Daughters of Vermont" and the "Sons of Vermont" did much to make the children happy during their stay in Boston. Owing to the prevalence of contagious diseases in Boston, it was deemed inadvisable to have the teachers come into the hospital daily to keep up the school work as had been done before. In spite of every precaution, however, the department sustained its first loss of life, for one of the children contracted scarlet fever three days after operation and died from an embolus. Forty-one cases in all were operated upon.

*Including operations performed on some non infantile paralysis cases.

at Kimball Cottage, the remaining 30 cases in Vermont, New York, or Boston, and the results of the year's work were wholly satisfactory.

In 1921, an addition to the work was made which seems to have been as important as any previous extension of activities. A careful study of the patients under the supervision of the department showed that while the greater number of adults were self-supporting, nevertheless there were a few who, because of their disability and isolation from suitable work, were unable to contribute materially to their own support. In September, 1921, a department of vocational training was started under the supervision of Miss Margaret B. Ives. This was done because there were two classes of cases who were found to be in need of such help. The patients who had been in the hospital and by operations and other treatment had been improved in their range of activities, on their return home became restless and unhappy because of their isolation and disability and it was obvious that if the best physical and mental results were to be obtained from these cases, some occupation must be provided. In a second class where the patient was isolated by his disability or perhaps lived in the mountains away from much contact with people, it became evident that some outside interest would be of great help in stimulating ambition, especially if it offered any hope of making the patient financially independent. This latter aspect,—the fact that a man practically helpless could earn a certain amount of money,—has proved most stimulating psychologically and physically. The patients are visited by their teacher at the outset who shows them the various possibilities in the way of making articles and instructs them in the one that they select. She visits them at frequent intervals to see that the standard of work is kept up and stimulates them to further efforts in improving their work or becoming independent in the matter of designing, etc. This department finds suitable

work for the patient, gives instruction free, furnishes raw material to be paid for when the products are sold, and finds a market for the articles made. The handicrafts taught are weaving, rug-making, toy-making, basket-making, and various types of needlework.

The results of this branch of the work have been most satisfactory, and the interest and appreciation shown have proved it worth the undertaking. Aside from the economic value of enabling these remade individuals to discharge their debt to the community, the moral effect of this work on the patients themselves can scarcely be over-estimated, for through it they have acquired independence and self-respect and have become useful citizens.

During 1922 no changes of any significance were made in the work of the department. Dr. Ober and I held a series of clinics in nine centers in August, and Miss Weisbrod held clinics in the same centers in May and October. At the clinics in 1922 very few operative cases presented themselves, a situation in great contrast to the conditions of the earlier clinics. It would seem to be that not only have the operative cases been taken care of, but the persistent following up of the cases and holding them to treatment have resulted in preventing, in a large measure, the severe deformities requiring operative treatment.

STATISTICS

Throughout these statistics cases of infantile paralysis, in which the onset of the disease occurred at least a year previous to the date of first examination by the Department for the After-Care of Poliomyelitis, are called "old cases." Cases which were examined within a year of the date of onset of the disease are called "recent cases."

The work began at the termination of a large epidemic throughout the state. At the clinics of the first year 243 cases were admitted, the largest number for any single year.

Two-thirds of these cases were poliomyelitis cases of the recent epidemic, but there were also 70 cases of long standing paralysis, showing the need of such work without an epidemic.

In the next year, 1916, there were only 42 admissions. Over half of these—24 cases—were recent cases, left-overs from the 1914 epidemic and imported cases from the larger 1916 epidemic in other states.

In 1917 the number of cases rose again, the total being 119. The large number of new cases—79—came almost entirely from the Montpelier district where there was a very severe but very local epidemic.

In 1918 the work was practically at a standstill, and only 14 new cases were recorded.

During the years 1919 and 1920 there was an entirely new distribution of cases, and one which might be regarded as the normal for an established work in years when there were no epidemics. In each year there was a small number of new cases, and a considerable number of old cases largely from the epidemics of 1914 and 1917.

TABLE 1

NUMBER OF POLIOMYELITIS CASES ADMITTED TO CLINICS
1914-1921

<i>Year of First Attendance</i>	<i>Old Cases</i>	<i>Recent Cases</i>	<i>Total</i>
1914 and 1915	70	173	243
1916	18	24	42
1917	40	79	119
1918	8	6	14
1919	54	15	69
1920	62	6	68
1921	38	41	79

In 1921 the incidence of the disease again increased, and is shown in the increase of recent cases admitted to the clinics.

The total number of cases of poliomyelitis admitted to the clinics to January 1, 1922, was 634. Of this number 54 per cent, or 344 cases, were cases which the department was given the opportunity to treat within one year of the onset of the disease. Forty-six per cent, or 290, were cases where the paralysis had been present for a year or more before the treatment was started.

TABLE 2

YEAR OF ONSET OF ALL POLIOMYELITIS CASES SEEN BY
THE DEPARTMENT TO JANUARY 1, 1922

<i>Year of Onset</i>	<i>Number of Cases</i>
1840	1
1880-1889	3
1890-1899	10
1900-1904	13
1905-1909	26
1910	30
1911	29
1912	10
1913	18
1914	207
1915	27
1916	60
1917	104
1918	10
1919	18
1920	6
1921	35
Date of onset not known	27
Total number of cases	634

TABLE 3

SUMMARY OF INACTIVE CASES OF POLIOMYELITIS
TO JANUARY, 1921

	<i>Old Cases</i>	<i>Recent Cases</i>	<i>Total Cases</i>
No treatment prescribed at first clinic attended*	4	35	39
Refused treatment	16	22	38
Private cases	1	0	1
Moved from state	20	27	47
Preferred osteopathic or chiropractic treatment	4	4	8
Died	5	11	16

Cannot be located	9	15	24
Cases dropped	5	12	17
Referred to New York Commission	1	5	6
No further treatment needed	14	34	48
All cases	79	165	244

*Includes abortive cases, cases showing no paralysis, and cases which were seen but once by the department.

In 1921, when this study was made, 244 cases of poliomyelitis which had previously been seen at clinics were no longer under active treatment. Of this number, 39 had not needed any treatment when first seen at the clinics, 35 of these being recent cases not showing any paralysis at the first examination. Thirty-eight cases had continually refused treatment, but the Department still carried many of these cases on its inactive files, hoping to help them later. Sixteen of the number had died, and 6 had been referred to the New York Commission. Seventeen cases had been dropped because of family conditions and lack of cooperation. No further treatment was needed by 48 cases, practically all of whom had been considered "normal" or "practically normal" by the attending surgeons.

Table 4 was compiled in 1921, and is a consideration of 380 cases. They include recent and old infantile paralysis cases which were admitted to clinics before January, 1920, and treated regularly by the Department.

Of the 380 cases, 155 were old and 225 were recent cases. Of the old cases 85, or 55 per cent, were considered to have followed treatment regularly during their supervision by the Department. That means that they did the prescribed exercises regularly, wore the apparatus advised, and underwent operations without long delay. Of these patients only two failed to improve, and at the time this was written these two were in approximately the same condition as they were at the time of admission. Thirty-nine per cent fol-

TABLE 4

NUMBER AND PERCENTAGE OF OLD AND RECENT CASES OF POLIOMYELITIS WHICH IMPROVED, REMAINED THE SAME, OR LOST, SINCE DATE OF ADMISSION, ACCORDING TO THEIR REGULARITY IN FOLLOWING THE TREATMENT PRESCRIBED

	Old Cases					Recent Cases				
	Improved	Remained the same	Lost	Total cases	Per cent	Improved	Remained the same	Lost	Total cases	Per cent
Cases following the treatment regularly . . .	83	2	0	85	55%	133	1	1	135	60%
Cases following the treatment irregularly .	34	25	1	60	39%	47	31	2	80	36%
Cases not fol- lowing the treatment ..	0	7	3	10	6%	1	5	4	10	4%
All cases treat- ed	117	34	4	155	100%	181	37	7	225	100%
Per cent of all cases	76%	22%	2%	—	—	81%	16%	3%	—	—

This table covers cases admitted previous to January 1, 1920, and actively treated by the Department.

lowed treatment irregularly; that is, they did their exercises only fitfully, wore their braces only part of the time, or delayed in having the advised operations. Nevertheless, 34, or more than half of the cases improved, and only one case became worse. The majority of the cases who did not follow treatment at all were cases which continually refused operation. In none of these cases was there any improvement, but only three of the ten actually lost ground.

Of the 225 new cases, 133, or 60 per cent followed treatment regularly, and of this number all except 2 gained. The only case which lost was a very light case which followed treatment regularly for several years, missed one year of clinics (1918), and in 1920 was found to have developed a slight cavus and claw foot, probably from overuse. The cases that followed treatment irregularly numbered 80, or 36 per cent; but more than half of these improved, 5 were the same in 1921, and 4 had lost.

In comparing the improvement of the old and recent cases, it will be seen that 81 per cent of the new cases gained, as contrasted with a gain in 76 per cent of the old cases. That the gain is greater in the new cases is due to the fact that the natural tendency of the disease is to improve, and it is well known to be more amenable in the first two years than at any other time. One remarkable thing shown by the figures is the large number of old cases which improved, for, while in some of the cases the duration of the paralysis had been short, in many instances it had been present for from 10 to 20 years. To a large degree, of course, this improvement was due to the performance of successful operations, but the figures also show that another class of case existed which was not operated on and yet improved late in the history. The improvement in this latter group was undoubtedly attributable to the fact that these cases had not had proper treatment since the onset of the disease and many had been practically neglected. The point should be

stressed, however, that improvement is possible in cases of many years' duration.

TABLE 5

NUMBER AND PERCENTAGE OF CASES OF POLIOMYELITIS
HAVING UNDERGONE, HAVING UNDER CONSIDERATION,
OR HAVING REFUSED OPERATIONS ADVISED BY
THE DEPARTMENT

	<i>Number Per cent</i>	
Cases operated on at the advice of the Department:		
1914-1919	62	
1920	49	
1921	44	
1922 (to July)	7	
	—	
	162	65
Cases in the hospital	13	5
Cases considering operations, the performance of which has been advised	17	7
Cases refusing to have operations advised	55	22
Cases having died or left the state before opera- tion could be performed	3	1
	—	—
Total number of operative cases	250	100
Percentage of all cases of poliomyelitis treated at the clinics which were operative cases ..	47.4%	

From these figures it will be seen that of the cases for which operation was deemed necessary, 22 per cent refused operative permission, 7 per cent were considering having the operations but had not made up their minds at the time these figures were compiled, and 1 per cent had died or left the state before operation could be performed.

Whether or not this situation would be found to exist in other localities cannot be decided, but in Vermont the experience has been that as the communities became better educated in the matter of operations and hospital treatment the percentage of cases willing and glad to go to a hospital and be operated on constantly increased. It was found in Vermont that no influence in the community was as powerful in diminishing the opposition to operative treatment as the return of successfully operated children to their homes,

and in localities where this occurred the opposition practically disappeared.

TABLE 6

NUMBER AND PERCENTAGE OF OLD AND RECENT CASES OF POLIOMYELITIS FOR WHICH EXERCISES WERE AND WERE NOT PRESCRIBED

	<i>Old Cases</i>		<i>Recent Cases</i>		<i>Total Cases</i>	
	<i>No.</i>	<i>%</i>	<i>No.</i>	<i>%</i>	<i>No.</i>	<i>%</i>
All cases admitted before Jan., 1920, and actively treated by the department	155	100	225	100	380	100
Cases for which exercises were not prescribed	51	33	31	14	82	22
Cases for which exercises were prescribed .	104	67	194	86	298	78

NUMBER AND PERCENTAGE WHICH FOLLOWED EXERCISES REGULARLY, IRREGULARLY, OR NOT AT ALL

	<i>Old Cases</i>		<i>Recent Cases</i>		<i>Total Cases</i>	
	<i>No.</i>	<i>%</i>	<i>No.</i>	<i>%</i>	<i>No.</i>	<i>%</i>
All cases for which exercises were prescribed .	104	100	194	100	298	100
Cases following exercises regularly	64	62	121	62	185	62
Cases following exercises irregularly	23	22	56	29	79	27
Cases not following exercises at all	17	16	17	9	34	11

This table was compiled in January, 1921, and includes only cases admitted and actively treated by the Department previous to January 1, 1920. With regard to the cooperation to be obtained from the patients, from the best information at our disposal, Table 6 shows what really happens with regard to following out exercises at home.

In many cases the parents considered that the patients did the exercises regularly, when that would hardly have been the opinion of the supervisor. Some cases did exercises very regularly under supervision, but were very irregular later when there was no supervision. Cases which did not

do their exercises well before operation were very careful about them after operation. It was also noticed that, in general, parents who gave exercises faithfully admitted their usefulness, while those who became discouraged after a couple of weeks felt that the exercises were "of no earthly use," and often expressed the opinion that the patient got "all the exercise he needed playing outdoors." Ninety-two per cent of the parents or adult patients who gave exercises regularly believed that they aided.

TABLE 7

WALKING ABILITY OF CASES OF POLIOMYELITIS
BEFORE AND AFTER TREATMENT BY THE DEPARTMENT

	<i>Old Cases</i>	<i>Recent Cases</i>	<i>All Cases</i>
Number of cases unable to walk at time of admission	36	52	88
Able to walk in 1922	27	46	73
Unable to walk in 1922* ..	7	4	11
Number of cases having died by 1922	2	2	4
The reasons for inability to walk in the 11 cases were as follows:			
Continually refused necessary operations			6
Practically no power available in legs or arms			3
No ambition to attempt walking			1
1921 case not yet allowed to attempt to walk			1
Total			11

The above figures show that of the 634 cases of infantile paralysis admitted to the care of the Department previous to January, 1922, 88, or 14 per cent were unable to walk at the time of admission. In July, 1922, 11 of the 634 cases, or 1.7 per cent were still unable to walk. Of this number, 7 were unable to walk because of their own lack of cooperation. One case had not been allowed to attempt walking. Of the 634 cases this leaves only 3 which, under favorable conditions of treatment and cooperation on the part of the patient, were still unable to walk in 1922,—less than $\frac{1}{2}$ of 1 per cent.

TABLE 8

OCCUPATIONS OF ALL INFANTILE PARALYSIS CASES ADMITTED
BEFORE JANUARY, 1920, AND ACTIVELY TREATED
BY THE DEPARTMENT*

<i>Children</i>	262
Go to school regularly	220
Go to school for crippled children	17
Go to school irregularly	4
Do not go to school ¹	10
Too young to go to school	11
<i>Adults</i>	80
Earn their own living	69
Earn part of their own living ²	6
Do not earn anything ³	5
<i>Dead</i>	14
<i>Occupation not known</i>	24
<i>Total cases</i>	380

*This table was compiled in January, 1922.

¹Includes 4 children mentally deficient, 1 child deaf and taught at home, 2 children living too far from school and taught at home, 1 child to go to school for crippled children, and 2 children unable to walk because parents refuse to allow necessary operations.

²Working under direction of Miss Ives, the Vocational Supervisor, and will eventually be self supporting.

³Includes 2 patients unable to walk because they refuse necessary operations, 1 patient too severely paralyzed, 1 patient refuses to co-operate with Vocational Supervisor, 1 patient who does a small amount of house work.

The two preceding tables deal with the present condition, from an economic point of view, of all cases of infantile paralysis admitted to the care of the department previous to January, 1920. The figures show that it is exceptional for a child to be unable to go to school regularly on account of his paralysis, and that the majority of the adults are able to earn their own living.

Of the 80 adults whose occupations are known, there are only 11 who are not entirely self supporting. An analysis of those who are self supporting shows that they did not as a rule seek sedentary occupation, but indulged in all sorts of activities. It should also be noticed that nearly 20 per cent

of the group now engaged in active work were unable to walk when first seen.

TABLE 9
OCCUPATIONS OF INFANTILE PARALYSIS CASES
EARNING THEIR OWN LIVING
1922

<i>Occupations</i>	<i>Number earning living</i>	<i>Number which were unable to walk at admission</i>
Farming	17	2
Housework	11	2
Office work	11	3
Garage work	4	2
Salesmanship	2	1
Lumbering	2	0
Factory labor	3	2
Telephone operating	2	1
Shoe repairing	1	0
Sewing	1	0
Teaching	2	0
Studying and working way through college	2	0
Photography	1	0
Gardening	1	0
Machine work	1	0
Running saw mill	1	1
Working, but exact occu- pation not known	7	0
Total	69	14

SUMMARY

The plan for the after-care of poliomyelitis in Vermont has thus gradually expanded, until, from being in 1914 just a series of public clinics it is now a definitely organized department of the State Board of Health, functioning the year around and undertaking the supervision of every case of poliomyelitis in the state from the time the quarantine is lifted until the best possible recovery has been made.

The preceding statistics were compiled from the records, and from them it seems evident that the number of disabilities from poliomyelitis has been very greatly diminished; that many individuals are now wage-earners who otherwise

would not have been; and that, on the whole, the patients have been appreciative and cooperative. I do not believe that anywhere there will be found a group of cases of poliomyelitis where the end results are more satisfactory than in the group of cases under consideration. The scheme, originally beginning in such a small way, has itself indicated the lines along which its development should take place and each addition has been justified.

At present, some years after a serious epidemic, my impression is that the problem has resolved itself into one largely of after-care, as practically all of the cases are on a level at which they should remain stationary. Unless another epidemic occurs, few surgical problems will arise in these cases; but the continuance of these patients as active, useful individuals depends upon their being carefully and persistently followed up. In other words, from being chiefly a surgical problem, the work has become largely a problem of supervision and after-care.

ORGANIZATION, AIM, AND PLAN OF WORK OF THE AFTER-CARE DEPARTMENT FOR POLIOMYELITIS

By BERTHA E. WEISBROD*

THE After-Care Department undertakes the supervision of every case of poliomyelitis in the State of Vermont from the time the quarantine is lifted until the best possible recovery has been made. The cases divide themselves into two main groups: old cases, in which the onset was prior to the establishment of the department and in which varying degrees of paralysis or deformity are already existent; and new cases, those of recent onset in which treatment may be supervised from the beginning and paralysis and deformity to the greatest possible extent prevented. Each acute case which is reported to the Board of Health has the benefit of the highly specialized knowledge of the department which works in close cooperation with the attending physician, giving advice as to treatment and preventive work, and, as soon as the acute stage is past, assuming full charge of the after-care until as complete a recovery as possible is assured.

The department is affiliated with the Board of Health of the State of Vermont, which provides it with office space, and it is under the general direction of Dr. Robert W. Lovett of Boston. The personnel in Vermont consists of a director, in complete charge of the work itself and responsible for the organization and maintenance of its subsidiary activities; an assistant in the field work; a vocational teacher; and a secretary.

*Director, Division of Poliomyelitis After-Care, Vermont Department of Public Health.

Each year mid-summer clinics are held at nine centers in different parts of the state in charge of Dr. Lovett or his associate, Dr. Ober, with the assistance of the field workers. At these clinics each case is diagnosed by the physician in charge, the necessary treatment is prescribed, parents and local physicians are advised as to the best methods to be pursued, and the exact status of each case under the supervision of the department is ascertained and recorded at this time. Patients who require special examination before the time of the annual clinics may be taken to Boston where Dr. Lovett or Dr. Ober is always available for consultation. Subsidiary clinics are held in the spring and fall in charge of the director and her assistant. At this time improvement is noted, changes in treatment indicated, and apparatus adjusted.

After the clinical examinations a careful follow-up of the treatment in the homes of the patients is maintained by the field workers. Each of these workers is provided with a car which enables her to cover a large area without waste of time. During the winter months when the roads are impassable for automobiles, the necessity of traveling by rail greatly reduces the number of visits which can be made each month. In addition to home visits, some of the patients call at the office for consultation, for special examination, or for adjustment of apparatus. Through these frequent visits, both home and office, the workers are enabled to see that the prescribed treatment is carried out, to give muscle training exercises, to measure for and adjust apparatus, and otherwise to give careful supervision to the patients in their charge.

In the cases where the clinical examination indicates that an operation is advisable, it is the duty of the director to consult with the family of the patient and obtain the necessary consent. This often requires a certain amount of educational work before it can be accomplished. The director

then makes all arrangements for the operation, secures hospital accommodations, assumes full charge of the patient on the trip to and from the hospital, keeps the family informed of progress and when the operation is over and the patient back at home, sees that proper post-operative care is given. In order to secure the best possible care for its operative cases, the After-Care Department has found it advisable to maintain a hospital of its own in Boston during the winter where the patients may be kept under its direct supervision. The director of the department is required to secure this hospital, equip it, obtain the staff and after it is running to keep it under her general supervision. In addition to the cases treated in this hospital, there are many patients cared for in local hospitals, in the Children's Hospital in Boston, and in the Hospital for the Ruptured and Crippled in New York.

The department also has its own appliance shop where most of the apparatus used for the Vermont cases is made. This is located in Burlington at the University. The braces are made directly from the measurements taken by the field workers so that the delay and uncertainty of ordering by mail are eliminated.

Another part of the work of the After-Care Department is Ormsbee House, a school and home for crippled children, designed especially for their use and with every provision for their welfare. This school is intended for children whose homes do not offer the best conditions for successful treatment and is free to those whose parents are unable to pay for their care. Ormsbee House has its own superintendent and staff but is under the general supervision of the After-Care Department.

The department gives vocational training to those of its patients who are unable to contribute materially to their own support. This part of the work is carried on by a vocational teacher who provides materials at cost and markets

the finished articles until each patient is in a position to do this for himself.

In addition to the organization and carrying on of the work itself, the director is also responsible for the management of the office. There are, of course, innumerable details of office routine incidental to managing the various activities of the department, and all the data are recorded as clearly and simply as possible.

A file (with card index) contains the patients' cards in simple alphabetical order, according to the name of the patient and grouped geographically according to clinic. The patient's card consists of two parts; a name card containing all the essential facts with regard to the patient at the time of his admission, and an action card which contains the record of treatment. The name card has on the face the name of patient; name of parent or guardian, and address; name of attending physician; clinic attended; date of admission; onset of disability; diagnosis; and treatment prescribed. On the reverse side of this card is a list of essential facts which may be checked for statistical purposes and which will tell at a glance the status of the patient. The action card contains the record of visits made, treatment prescribed, results, etc. There is a cross-filing system which indicates which patients are in hospitals and which patients are having apparatus made or repaired. This card file is adequate for the ordinary daily routine, but in order to have more detailed information for the clinics, there is an additional file containing a folder for each patient and arranged according to the same alphabetic, geographic system and for which the same card index (which also serves as a mailing list) is used. Each of these folders contains a complete muscle chart for the patient and all papers relating to case history.

The vocational file is also a simple, alphabetical file con-

taining work cards for each patient with complete record of material issued, work finished, on hand, sold, etc., and action cards containing record of visits made by vocational assistant, supervision given, etc.

Besides the daily records made for the office itself, a detailed report is rendered monthly and a full report annually to the State Board of Health.

Through the various branches of its work, the department endeavors to give the best possible surgical treatment and physical care to its patients, to secure home conditions which will render a satisfactory recovery possible, and to establish the patient as a useful member of the community. This service is, of course, without charge and in the operative cases, where there are hospital or apparatus expenses to be met, if the patient is unable to pay the full amount, financial assistance is given. Beside carrying on the actual work, the department makes every effort to record all results accurately and to keep such records clear and immediately available, so that the statistics compiled therefrom may prove of definite value in the further pursuit of the work and may serve as a true basis of comparison for any future experiment along the same lines.

HISTORY OF THE WORK OF THE RESEARCH LABORATORY

By W. L. AYCOCK, M.D.

THE year 1914 will be memorable in the public health records of Vermont, not only because of the widespread and serious outbreak of poliomyelitis which swept the state in that year, but also because this dire event was responsible for the development of a form of practical philanthropy previously unknown in Vermont. An anonymous friend placed at the disposal of the State Board of Health a considerable sum of money to be used for the purpose of investigating the nature of this baffling malady and placing the benefits of the best possible methods of treatment within the reach of those afflicted with the disease. Dr. Caverly had called attention to infantile paralysis as a problem of distinct importance from a public health standpoint as early as 1894, when he recorded the first epidemic which occurred in this country. From that time until 1910, the disease did not reappear in epidemic form, although occasional cases were encountered in the state. In the meantime, the disease had become prevalent in neighboring states, especially during the years from 1907 to 1910. Upon the reappearance of the disease in Vermont in 1910, active measures were taken to guard against it. It was made a reportable disease and a systematic epidemiological study was begun by Dr. Caverly. His investigations, which were recorded in his annual reports and are reprinted in this book, provided an insight into the behavior of epidemic poliomyelitis which was invaluable in outlining the work to be undertaken under the gift which came to the state in 1914.

After conferring with Dr. Simon Flexner, Director of the Rockefeller Institute for Medical Research, and with Dr. Robert W. Lovett, Professor of Orthopedic Surgery in Harvard University Medical School, three distinct lines of work were undertaken: (1) the care of patients in the acute and chronic stages of the disease; (2) preventive work, consisting of study and investigations of cases and epidemics, assistance to physicians in diagnosis, and the regulation of quarantine; (3) experimental investigations of the nature, mode of transmission, treatment and prevention of the disease.

It was at once realized that the success of an organized effort of this kind would depend in a large measure upon the coöperation of practicing physicians of the state who are the first to come in contact with cases of the disease. A campaign was instituted for the purpose of bringing to the medical profession of the state the most advanced knowledge in regard to the diagnosis, treatment, and prevention of this disease. It should be remembered that at this time epidemic poliomyelitis was still a comparatively rare disease in most sections of the state. In pursuing this campaign the State Board of Health had the services of Dr. Francis R. Fraser of the Presbyterian Hospital of New York City. Meetings were held at different points in the state and everything possible was done to familiarize the medical profession with the disease. Much was accomplished in the way of stimulating interest in efforts to control the spread of the infection. Later, this portion of the work was incorporated with other departments of the State Board of Health whose work brings them in contact with physicians throughout the state. They provide information regarding the disease which would not be available otherwise.

That portion of the work which has to deal with acute cases, that is, the epidemiologic investigation, diagnosis and

treatment of the acute stage of the disease, and the experimental investigation of the infectious agent, has been carried on by the Research Laboratory of the State Board of Health. This work was organized by Dr. Harold L. Amoss of the Rockefeller Institute for Medical Research in 1914, and since that time it has been carried on under his supervision. In 1914 and 1915 the work was conducted by Dr. E. S. Towne; from 1915 to 1918 by Dr. Edward Taylor; during 1918 by Dr. Peter Noe; and since 1919 by Dr. W. L. Aycock. During his lifetime, Dr. Caverly was in close touch with the activities of this department and was always a source of inspiration to those who labored under his direction upon its difficult and sometimes discouraging problems. Since it was first put into operation, now nearly ten years ago, the original plan of the work has been maintained without essential changes—a tribute to the wisdom and foresight of its founder, Dr. Caverly.

The diagnostic facilities of the laboratory have been constantly available to all portions of the state, a fact which is of great value in checking the spread of the infection, in that it makes possible the recognition of even the more obscure cases, which is of prime importance in any campaign for the suppression of disease. In order that this service might be available for the examination of cases early in the course of the disease, a portable laboratory outfit which may be carried to the patient's home is used. Because of the size of Vermont a call to any part of the state is responded to within a day, usually by automobile, and the laboratory examinations are made at the bedside. Thus, by investigating cases or suspected cases very early in the disease, a splendid opportunity is afforded for carrying out uniform sanitary measures and treatment by special methods. It is by reason of this early contact with the acute case that the laboratory staff has been able to use as much convalescent serum as has been obtainable for the treat-

ment of cases. Thus, this portion of the work, which has been known as the "field work," brings the laboratory into contact with all the problems of the disease, and provides the basis for the research work.

Actual experience with the disease has pointed out many problems which have been the subject of investigation in the laboratory. In a general way, the experimental investigations have been directed toward the solution of the practical problems of poliomyelitis. This work has already served to fill in some of the important gaps in the knowledge of the disease. Some of the results of the laboratory investigations which have been published in various journals are reprinted elsewhere in this volume. Efforts have been made in various ways to produce serum active against the virus. Again, the possibility of producing a vaccine or immune serum by the use of pure cultures led to many attempts to cultivate the virus artificially. This work, which has thus far been unsuccessful, led in its turn to a series of experiments in which cultivation of the virus was attempted by reproducing as nearly as possible the conditions under which it multiplies when inoculated into monkeys. The virus was inoculated into culture media contained in collodion sacs, which were implanted into animals.

These methods failed to produce a more potent serum for the treatment of the disease, so attention was directed toward the methods used in the administration of convalescent serum, which has a destructive action on the virus. A study of the secretion and circulation of cerebrospinal fluid indicated that administration of serum by the usual methods does not insure the greatest action of the serum upon the virus. Furthermore, within recent years certain alterations have been experimentally produced in the circulation of cerebrospinal fluid which suggested possibilities in connection with the problem of securing a more adequate destruction of virus by serum. This work is still being carried on.

In a similar manner many other phases of the problem have been pursued. Many substances, chemical and biological, have been tested in attempts to find some destructive agent which could be applied to the prevention or treatment of the disease; methods for detecting carriers have been studied; and the mechanism through which the virus is able to set up infection has been investigated, along with the question of susceptibility to infection. In the hope that certain analogies between the virus of poliomyelitis and that of other diseases might point to a solution of some of the problems which have arisen, experiments which were not possible with this virus have been carried on with other infectious agents which bear a similarity to this virus. Many of the experiments have given valuable results, while others have failed entirely; still other questions studied in the light of our present knowledge will require repetition as the progress of the sciences of bacteriology and pathology make available improved methods for carrying out such investigations.

Thus the work has progressed, the practical side of the work serving its purpose toward prevention of the disease and the compilation of data obtained in the field continuously pointing to problems which are made the subject of investigation in the laboratory.

Any organized effort directed toward the eradication of epidemic disease and carried on in a manner calculated to be for the public welfare necessitates certain restrictions on the ordinary pursuits of individuals and communities which may be a matter of serious inconvenience to them. Thus the work of this organization, in the effort to lessen the occurrence of poliomyelitis, calls upon the physician, individual and community alike, to take part in a way which often amounts to a great sacrifice. On every hand there has been encountered a willingness to undergo any amount of hardship to lessen the menace of this dread disease.

THE CARE AND TREATMENT OF POLIOMYELITIS IN VERMONT*

W. L. AYCOCK, M.D.

I HAVE been invited to speak here of the care and treatment of Acute Anterior Poliomyelitis. I assume that what is desired at a conference of this sort is a discussion of these features of the disease as they apply to the general problem rather than to the individual case.

It is an obvious truism that the control of communicable diseases is of vital importance to the community in which such diseases occur. A disease which can be transmitted from one individual to others by any means whatsoever is a matter which concerns not only the individual who has it, but also those to whom it might be transmitted. Furthermore, all diseases which are transmitted by natural means are capable of reaching epidemic proportions, in which case they become a menace to a greater number of people. It follows then that the appearance of a communicable disease is a source of danger to all, and, realizing this indication of a possible widespread outbreak, the people of a community make certain attempts to avoid the disease. This interest which a community takes in its state of health is nothing new. As far back as history goes we find evidence that communities have made efforts to rid themselves of epidemic diseases. The cry "Unclean" was one of the earliest quarantine measures. Its success was due to its universal rigid enforcement and to this day it stands out as one of the most thorough public health measures. Using a similar method we could quickly drive out any communicable disease with which we have to deal today. In a few instances, the houses

*Read before the Conference of the Vermont State Medical Society and the Public Health Workers, Jan. 11, 1922.

in which the dead have lain have been burned down to prevent the diffusion of the contagion. There was a time when ships were sunk to destroy the supposed contagion on board. Before the days of vaccination the people concerned themselves with the problem of smallpox to the extent that they had themselves inoculated with it in order to develop the disease and have it over with at some convenient time. Indeed a remnant of this primitive attempt at handling the question of communicable diseases persists in some quarters. There are still found those who like to expose their children to some of the common infectious diseases at some time when it is most convenient to get over with what they consider the inevitable. These represent the attempts of the laity in earlier times to handle their medical problems unaided.

With the development of the medical profession, the problem has been turned over to them, and our communities now look to us for protection against ravaging diseases. With the increasing knowledge on the part of the public that many diseases are preventable, for which they are indebted to the medical profession, this demand is growing. How are we to meet it?

In order to control or eradicate a communicable disease, its behavior must be understood. Its cause must be known, it must be capable of identification, the manner in which it leaves the sick, spreads, and gains its footing in the healthy must be understood. When these things are learned, the remedy is to be sought. Something which will cure the sick must be found. A way to prevent its leaving the sick, spreading, or gaining entrance into the healthy individual must be contrived. These are the things which must be discovered before the demands for protection can be satisfied and this knowledge can only be acquired by the collection and correlation of observations and by testing possible hypotheses in the experimental laboratory.

It is in this manner that the great strides in the prevention of epidemic diseases have been made. The rapid disappearance of malaria is following the discovery of the mosquito as its carrier, and the same is true of yellow fever. The eradication of smallpox depends only upon the thoroughness with which we use the methods which have been placed at our disposal. Typhoid fever has quickly yielded to the methods which have been developed for the control of the spread of its organism and widespread vaccination against it. Rabies has succumbed to Pasteur's wonderful discovery. There are many other examples, all the product of organized efforts to collect and correlate observations and to test various hypotheses experimentally.

There remain unsolved many other similar problems, notable among them tuberculosis, the pneumonias, venereal diseases, and the infectious diseases of childhood. Poliomyelitis is only one of them, and I wish to have you consider with me the present state of our knowledge of this disease and the principles underlying the efforts which are being made in Vermont to check its ravages.

Since the first epidemic of considerable size in this country, which occurred in Vermont in 1894, and which was described in medical literature by the late Doctor Charles S. Caverly, hardly a year has passed during which poliomyelitis has not appeared within this state. Since 1907 it has been on the increase in this country and Vermont has borne its share of this increase. In 1921, there were reported in the United States over 5,000 cases and 60 of these were in Vermont.

From the standpoint of the individual, poliomyelitis means death in from 10 to 20 per cent of cases, permanent total disability in about 5 per cent, and more or less crippling in over 50 per cent.

From the standpoint of the community, every initial case in a given section (based on Vermont statistics for 1921)

has been followed within three weeks by an average of three other cases in the same vicinity. The first appearance of the disease in any locality is thus an extremely important event because it is a forecast of other cases.

These features of the disease in addition to the fact that medical science cannot, with its present knowledge, prevent or cure the paralysis which results from it, are the strongest arguments for using whatever knowledge we now possess to prevent the disease and for adding to that knowledge by every possible means.

Poliomyelitis is an infectious and communicable disease which is caused by the invasion of the central nervous organs by a minute filterable organism.

The virus exists in the central nervous organs and upon the mucous membrane of the nose and throat and of the intestines in persons suffering from the disease either in its paralytic or abortive form. It is also present upon the mucous membranes of the nose and throat of healthy persons who have been in intimate contact with acute cases, and such contaminated persons, without falling ill themselves, may convey the virus to other persons who develop the disease.

The virus is known to leave the infected human body in the secretions of the nose, throat, and intestines, and to escape from the contaminated healthy person in the secretions of the nose and throat.

The chief mode of demonstrated conveyance is through the agency of human beings, the conveyors being the persons ill with the disease in any of its several forms and healthy persons contaminated with the virus through contact with the sick.

While other modes of infection may operate, the main avenue of entrance into the body seems to be by way of the upper respiratory mucous membrane.

This comprises, in brief, our knowledge of the manner

in which poliomyelitis spreads, and it has been acquired through the collection and correlation of observations and by the testing of various hypotheses in the experimental laboratory. Upon this knowledge is based the means of control which we can now bring to bear upon the disease, namely, the prompt discovery and isolation of those sick with any form of the disease and the sanitary control of those who have been in contact with the sick.

More remains to be learned about the disease before we can fulfill the demand of the public for protection against it. There exists, at present, no safe method of preventive inoculation or vaccination and no practical method of specific treatment. How can the disease be detected earlier in its course? How can the abortive case be detected with certainty? Are there other modes of transmission which have not yet been demonstrated? How can a healthy carrier be detected and how long may the virus be carried? Is there a way to prevent the transmission of the disease by removing the virus from the nasal passages or can the nasal mucous membrane be protected from the entrance of the virus? How can the organism responsible for the disease be artificially grown so that it will be available for experiments looking toward the production of a serum or vaccine? If, and when, it is so cultivated, can a serum or vaccine be made? If no way can be found to prevent the disease, what can be done to lessen its disastrous results? What is the direct cause of the injury to the nerve cell which gives rise to the paralysis? Can anything be done to lessen or prevent this injury?

In the answers to these questions, in addition to the most thorough application of our present knowledge of the disease, lies the ultimate solution of the problem.

The essentials of this research are: the earliest possible diagnosis of every case that appears, the collection and compilation of information regarding the date, location, the

surrounding conditions, and the significance of the relation of every case to other cases in the same or distant localities, and the testing of any hypothesis which this information points to in the laboratory. Success depends upon the individual practicing physician. That he may better perform his task, he must be provided with whatever aid he does not possess in order to make the earliest possible diagnosis and to collect the required information.

These are the principles underlying the work which is being done in Vermont. Thanks to the work of Dr. Caverly and the generosity of an anonymous donor, the practicing profession in Vermont now constitutes a group better able to recognize and handle the problem of poliomyelitis and to increase our knowledge of it than exists in any other state. This is the best guaranty our state can have for its safety.

NEUTRALIZATION OF THE VIRUS OF POLIOMYELITIS BY NASAL WASHINGS*

By HAROLD L. AMOSS, M.D.

AND

EDWARD TAYLOR, M.D.

(From the Research Laboratory† of the Vermont State
Board of Health, Burlington.)

THE occurrence of the infectious microorganism of poliomyelitis—the virus, so called—in the mucous membranes of the nasopharynx and in their secretions is now firmly established.^{1,2} Not only may the virus be demonstrated by inoculation tests during the acute period³ of the disease, but it is known to persist there in some cases for many months after convalescence^{4, 5} and, conversely, it has been detected in certain instances in the washings from the nasopharynx of healthy persons who have been in intimate contact with the acutely ill.⁶ Finally, the fact has been determined by experiment that when the virus is introduced directly into the central nervous tissues wherein it multiplies, it also appears in the mucous membranes of the nose and throat. These facts indicate that the nasopharyngeal mucous membranes play an important part in the pathology of epidemic poliomyelitis; and the weight of opinion today is to the effect that the ingress and egress of the virus take place by way of these structures.

*Reprinted from *The Journal of Experimental Medicine*, April 1, 1917, Volume XXV, No. 4, pp. 507-523.

†Maintained by a special fund privately donated.

1. Flexner, S., and Lewis, P. A., *J. Am. Med. Assn.*, 1910, liv, 535.
2. Landsteiner, K., Levaditi, C., and Pastia, C., *Semaine méd.*, 1911, xxxi, 296.
3. Flexner, S., and Clark, P. F., *J. Am. Med. Assn.*, 1911, lvii, 1685. Landsteiner, Levaditi, and Danulesco, *Compt. rend. Soc. biol.*, 1911, lxxi, 558.
4. Lucas, W. P., and Osgood, R. B., *J. Am. Med. Assn.*, 1913, lx, 1611.
5. Kling, C., Pettersson, A., Wernstedt, W., and Josefson, A., *Communications Inst. méd. État a Stockholm*, 1912, iii.
6. Flexner, S., Clark, P. F., and Fraser, F. R., *J. Am. Med. Assn.*, 1913, lx, 201. Kling, C., and Pettersson, A., *Deutsch. med. Woch.*, 1914, xl, 320.

One of the most important questions arising out of the data presented above is that relating to the so-called healthy carriers of the virus, and for two main reasons. The healthy carriers may be the means of transporting the virus to other persons less resistant who may develop poliomyelitis; or the carrier, healthy when first contaminated, may subsequently develop the infection. At present the means at our disposal for studying the subject of virus carriers are so imperfect that no adequate notion of their number and distribution can be obtained. As long as the inoculation of monkeys with the washings from the nasopharynx must be relied upon to furnish this information, complete knowledge cannot be acquired.

There is, however, another fact which may prove to be significant. Assuming that during the prevalence of epidemics, many persons become contaminated with the virus, the question arises whether this condition need necessarily be either a menace to the contaminated person himself or to others. The answer to this question may lie in the reaction of the secretions of the nasopharyngeal mucous membranes to the virus present upon them. It is possible that in one person the secretions do not exercise a harmful action on the virus, while in another they do. This injurious action upon the virus may be of the nature of a protection to the individual contaminated as well as to the public in general.

It has often been observed that washings made from the nasopharynx may be ineffective when introduced into monkeys, and the lack of power to cause infection has been attributed to insufficient quantity or low infective power of the virus believed to be contained in the secretions removed. No note has been taken of the possibility that the washings are ineffective because the secretions of the mucous membrane are destructive or neutralizing to the virus of poliomyelitis. While this possible action may affect the inoculation tests in cases of acute poliomyelitis, it would be far

more likely to be operative in the supposed carrier because of the small amount of virus and the probability of diminished virulence in the latter. Because of these considerations, a series of experiments was carried out to determine (1) the smallest quantity of a standard virus which can be detected in washings, and (2) the action of the washings of different persons upon the virus itself.

Reference has already been made to the fact that nasal washings, of contacts especially, have in a few instances produced poliomyelitis when injected into monkeys. As the virus obtained directly from human beings possesses low virulence for monkeys, and is injected greatly diluted, the small number of successful inoculations is significant. Tests were made to determine the effect of concentration of washings on the activity of the virus. Amounts of virus which would certainly produce the infection if injected directly were added to a filtered washing fluid obtained from persons not having been exposed to the infection. The mixtures were separately reduced to small volume *in vacuo* at low temperatures and injected into monkeys. The results obtained were variable, for reasons which at first were not obvious, but the tests nevertheless showed that the filtered virus in certain amounts may withstand concentration in washing fluids without losing entirely its infective power.

EXPERIMENTAL

Experiment 1.—The nasal cavities of a normal adult, H. L., were rinsed thoroughly with 50 cc. of distilled water. The collected fluids were passed through a Berkefeld filter and 0.2 cc. of a Berkefeld filtrate of a 5 per cent suspension of poliomyelitic brain was added. The mixture was reduced *in vacuo* at 37°C. to 4 cc. and injected intracranially, under ether anesthesia, into a *Macacus rhesus*. The monkey became partially paralyzed on the 10th day, completely prostrated on the 12th day, and died on the 14th day. Typical lesions of poliomyelitis were present.

Experiment 2.—The washings of a normal adult, W. T., were obtained in the manner described above. To the fil-

tered fluid was added 0.5 cc. of a Berkefeld filtrate of active virus. The mixture was concentrated at 37°C. *in vacuo* and injected intracranially, under ether anesthesia, into a *Macacus rhesus*. The monkey remained well. As the result of a later protection test the monkey died of poliomyelitis after an appropriate injection of potent virus.

Filtration through Berkefeld or other porcelain filters is undertaken to remove the bacteria always present in the nasal and buccal secretions. But the bacteria can be either killed or their multiplication inhibited by certain antiseptic chemicals which affect to a less extent the virus of poliomyelitis. Thus 0.5 per cent carbolic acid destroys pyogenic bacteria in tissues and leaves the virus intact. Experiments also showed that ether acted more severely on the ordinary bacteria than on the virus. In order, therefore, to obviate any loss of virus which might result from its retention by the filters, ether was employed to sterilize the washings. Preliminary tests showed that contact of ether for 20 hours with the virus contained in an emulsion of the spinal cord does not destroy it. The test made with washings indicates that while a shorter exposure may not kill all the bacteria, yet they are so greatly diminished that no ordinary infection is produced on inoculation into monkeys. And yet, as the experiments which follow show, while the filtrate contained in 0.8 per cent salt solution is active after the ether treatment, that mixed with the nasal washings is ineffective. The ineffectiveness at first believed to have been due to injury of the virus by the ether or too great dilution of the fluid inoculated, is now probably explicable in other ways.

Experiment 3.—1.8 cc. of a Berkefeld filtrate of active virus were added to 4.2 cc. of isotonic sodium chloride solution and 1 cc. of chemically pure ether. The mixture was shaken for 20 hours at room temperature. The ether was allowed to evaporate and 1 cc. of the remaining mixture, representing 0.3 cc. of virus filtrate, was injected intra-

cranially, under ether anesthesia, into a *Macacus rhesus*. The monkey was almost prostrate on the 7th day, completely prostrate on the 8th, and etherized when moribund on the 10th day. The lesions were typical.

Experiment 4.—To 100 cc. of nasal washings from two normal adults was added 1 cc. of a Berkefeld filtrate of mixed virus, 0.1 cc. of which produced paralysis in the control monkey in 7 days. 5 cc. of chemically pure ether were added to the mixture and the whole was shaken for 20 hours at room temperature. The ether was allowed to evaporate, and 2 cc. of the mixture were injected intracerebrally and 98 cc. intraperitoneally, under ether anesthesia, into a *Macacus rhesus*. The monkey remained well.

Berkefeld filters withhold even very minute particles in greater amount when they are contained within a viscid or glutinous liquid. All the washings contain mucus; hence a procedure was adopted to modify the mucin so as to avoid this difficulty without at the same time injuring unduly the virus itself. The procedure consists in treating the washings with sodium bicarbonate, filtering, and then concentrating *in vacuo* at 37°C. The virus is little injured. When 0.1 cc. of a filtrate, which is on the limits of a minimum lethal dose, is used, the resulting concentrated fluid may be ineffective; when 0.2 to 0.3 cc. is employed infection results. The next protocol in which 0.3 cc. of filtrate was used is an example of the method, but identical effects were obtained with 0.2 cc.

Experiment 5.—The nasal cavity of a normal adult was thoroughly syringed with 50 cc. of sterile distilled water. 0.3 cc. of a Berkefeld filtrate of virus was added, and, after thorough mixing, 0.25 gm. of dry sodium bicarbonate was added and the fluid shaken for 20 minutes with beads. After centrifugation at high speed for 3 minutes the fluid was decanted and passed through a Berkefeld candle V. The precipitate was washed and filtered through the same candle. The mixture of the filtrates was reduced *in vacuo* at 36°C. to a volume of 2 cc., which with rinsing water was transferred to a collodion sac and dialyzed for 1 hour.

Under ether anesthesia, a *Macacus rhesus* received half

(3.5 cc.) of the resulting liquid into the left, and the other half into the right cerebral hemisphere. No symptoms were observed until the 6th day when the monkey became ataxic and excitable. The monkey was prostrate on the 8th day and died on the 14th day after injection. Typical microscopic lesions of poliomyelitis were present.

The treatment of the washings with sodium bicarbonate renders the effect of the inoculation certain when the use of larger quantities of the virus with washings alone fails to confer infection. The probable cause of the discrepancy has become apparent only after a more minute study of the properties of the nasal washings; but that the sodium bicarbonate acts either by allowing more virus to pass through the filter or by removing certain inhibitory influences exerted by the washings is directly indicated.

INACTIVATING EFFECTS OF NASAL SECRETIONS UPON THE VIRUS

The results of the preceding experiments, which contained obvious discrepancies, suggested a closer study of the secretions of the nose and pharynx from the standpoint of a possible inhibiting or neutralizing action on the virus of poliomyelitis. For this purpose a variety of persons was studied; some were suffering from acute poliomyelitis, and the others were apparently normal individuals.

The nasopharynx was rinsed with double distilled water and the washings were fractionally sterilized by heating to 60°C. for 3 successive days. Each person's specimen was handled separately. In earlier experiments, in order to economize animals, the washings of several persons were often mixed. It now seems not improbable that discordant results follow this procedure. The virus employed was obtained by filtering a 5 per cent suspension of glycerolated poliomyelitic monkey spinal cord. To each 30 cc. of the washing 7.5 cc. of the filtered virus were added. The mix-

ture was then incubated at 37°C. for 24 hours. Control mixtures of virus and distilled water were subjected to the same incubation. Each cubic centimeter of the mixtures then contained 0.2 cc. of the filtrate, or at least two minimum lethal doses of the virus. The results of the first tests are given in Table I.

TABLE I.

Inactivating Effects of Nasal Secretions upon the Virus

<i>Date</i>	<i>Virus No.</i>	<i>Dose of virus filtrate</i>	<i>In contact with nasal washings from</i>	<i>Method of sterilizing nasal washings before addition of virus</i>	<i>Temperature at which virus plus nasal washings were incubated for 24 hrs.</i>	<i>Result</i>
1916		cc.			°C.	
Feb. 16	32	0.2	Baby C.; age 3 yrs. Acute stage of poliomyelitis	Heated 1 hr. at 60° C. on 3 successive days	37	Monkey died. Typical poliomyelitic lesions
Mar. 11	32	0.2	W. T., normal adult; age 39 yrs.	"	37	Monkey remained well
June 2	48	0.2	C.A.R. and L.M. McK. (mixed), normal adults	"	37	"
" 2	48	0.2	"	"	4	"
" 2	48	0.2	H. E. G., normal adult	"	37	"

The results of this experiment suggest that the nasal washings of a person suffering from acute poliomyelitis may exercise no restraining influence upon an active virus, while those from healthy persons, under identical conditions of preparation, inhibit its activity.

The next experiment comprised tests on the nasal washings of eight apparently healthy persons. The results are recorded in Table II. At first sight it appears that of the eight specimens of washings, six possessed inhibiting properties and two did not. The question arose as to whether

examination by a rhinologist, who would be unaware of the experiment, would disclose any differences in the nasal mucous membranes. These examinations, consented to by

TABLE II.

Inactivating Effects of Nasal Secretions of Adults

<i>Date</i>	<i>Dose of Berkefeld filtrate of Virus</i> 48	<i>In contact with nasal washings from</i>	<i>Method of sterilizing nasal washings before addition of virus</i>	<i>Temperature at which virus plus nasal washings were incubated for 24 hrs.</i>	<i>Result</i>
1916 Apr. 26	cc. 0.2	M. J. P., normal adult	Heated 1 hr. at 60° C. on 3 successive days	°C. 37	Monkey remained well
" 26	0.2	E. S. S., normal adult	"	37	"
" 26	0.2	V. H. S., " "	"	37	"
" 26	0.2	C. A. R., " "	"	37	"
" 26	0.2	L. M. McK., normal adult	"	37	"
" 26	0.2	G. H., normal adult	"	37	"
" 26	0.2	J. P. B., " "	"	37	Monkey died Typical lesions
" 26	0.2	H. E. G., " "	"	37	"
" 26	0.2	Control (sterile water)	"	37	"
" 26	0.2	Control (isotonic salt solution)		37	Monkey remained well

the persons, were kindly undertaken by Dr. M. C. Twitchell. His report is summarized in Table III. The only comment which the examination calls for is that while the anatomical condition of the nasal and adjacent mucosas in the six persons whose secretions contained inhibiting, inactivating, or neutralizing substances were normal, those of the other two were more or less pathologic. Just what the relation of this fact is to the effects of the secretions on the virus can only be surmised; but the test demonstrates that the secretions may frequently inhibit the action of the virus in monkeys.

TABLE III.

Results of Rhinoscopy of the Subjects Recorded in Table II.

<i>Case No.</i>	<i>Age yrs.</i>	<i>Report</i>
1 (M. J. P.)	23	Normal nasal respiration; no discharge; no history of colds. Mild hypertrophic rhinitis. Septum deflected slightly to left with large horizontal ridge.
2 (E. S. S.)	24	Normal nasal respiration; no discharge; no throat symptoms; no history of colds. Mild hypertrophic rhinitis; vocal bands red (subacute laryngitis).
3 (V. H. S.)	24	Normal nasal respiration; no discharge; no throat symptoms; no history of colds. Mild hypertrophic rhinitis. Two spurs on left side of septum.
4 (C. A. R.)	20	Normal nasal respiration; no discharge; no throat symptoms; no history of colds. Mild hypertrophic rhinitis; septum deflected, half closes right nasal cavity.
5 (L. M. McK.)	22	Normal nasal respiration; no discharge; no throat symptoms; no history of colds. Mild hypertrophic rhinitis; small ulcer on left side of septum; horizontal ridge on right side of septum.
6 (G. H.)	21	Normal nasal respiration; no discharge; no throat symptoms; no history of colds. Mild hypertrophic rhinitis. Acute pharyngitis of 3 days' duration.
7 (J. P. B.)*	22	Normal nasal respiration but easily obstructed when patient has a cold, especially left side of nose; secretion drops into throat on arising in the morning. Nose narrow; moderate hypertrophic rhinitis; large spur on right side of septum touches turbinate; small ulcer on left side of septum.
8 (H. E. G.)*	25	Nasal respiration interfered with, especially on right side; secretion drops into throat; frequent colds; has cold now. Septum deflected slightly to right; hypertrophic rhinitis; secretions found in right nasal cavity by anterior rhinoscopy. Acute pharyngitis and rhinitis.

*Remarks by Dr. Twitchell: "No. 7 shows the most marked chronic nasal trouble of all, and I should class it as moderate rather than severe. No. 8, at the time of examination, had an acute rhinitis and an acute pharyngitis. This to a certain extent obscures the findings in this case. Frequent colds are a marked feature in the history of chronic rhinitis. No. 8 is the only one giving this history. I should conclude that if a chronic rhinitis produces changes in the nasal secretions, No. 8 would be the one whose nasal secretions were the most changed."

The control tests (Table II) show that, under the conditions of the experiments, distilled water injures the filtered virus less quickly than isotonic salt solution, a fact possibly dependent upon the different osmotic conditions present in the two fluids. The inactivation of the virus through dilution by the washings and incubation at 37°C. would appear to be excluded by the results of the tests with the secretions and with the controls.

FLUCTUATIONS IN INACTIVATING PROPERTIES

Attempts were made to ascertain whether the action described is a constant or a variable property of the secretions. For this purpose washings were made at different times, sterilized by discontinuous heating at 60°C., and tested against 0.2 cc. of the filtrate which in control tests was determined to be potent. The results of these tests are given in Table IV.

TABLE IV.

Fluctuations in Inactivating Properties

<i>Date</i>	<i>Case</i>	<i>Condition</i>	<i>Dose of virus filtrate</i>	<i>Result</i>
1916			cc.	
Mar. 11	W. T.	Apparently normal	0.2	Neutralized
June 16	"	" "	0.5	"
July 12	"	" "	0.2	Failed to neutralize
Nov. 14	"	" "	0.2	Neutralized
Apr. 26	H. E. G.	Chronic rhinitis	0.2	Failed to neutralize
June 2	"	" " (improved)	0.2	Neutralized
July 12	"	Apparently normal	0.2	Failed to neutralize
Apr. 26	C. A. R.	" "	0.2	Neutralized
June 2	"	" "	0.2	"
Apr. 26	G. H.	" "	0.2	"
Dec. 18	"	" "	0.2	"
Apr. 26	L. M. McK.	" "	0.2	"
June 2	"	" "	0.2	"
Apr. 26	E. S. S.	" "	0.2	"
July 12	"	Acute coryza	0.2	Failed to neutralize

Of four tests with the secretions of W. T., three neutralized the virus; of three with those of H. E. G., only one neutralized it; of two with washings from C. A. R., G. H., and L. M. McK., respectively, all neutralized it, while in the case of E. S. S., one neutralized and the other did not. The animals that did not come down were subsequently determined to be susceptible to inoculation with the virus, so that the neutralization effects could not have been simulated by an excessive resistance on their part.

In addition to the tests described, which were conducted chiefly with adults, several were made with washings from children either healthy or suffering from poliomyelitis. The results are not wholly concordant. A larger series may possibly clear up the discrepancies.

Aug. 9, 1916. The washings of C. A., an apparently healthy boy, age 14, failed to neutralize 0.2 cc. of filtrate.

Oct. 23, 1916. The washings of R. J., age 8, taken during the acute attack of poliomyelitis, but after immune serum had been administered, neutralized 0.2 cc. of filtrate. A control monkey developed fatal, typical poliomyelitis.

Nov. 14, 1916. The washings of R. C., age 8, taken on the 15th day of the attack of poliomyelitis neutralized 0.2 cc. of filtrate. This patient had not been treated with immune serum. The control animal developed typical fatal poliomyelitis.

Feb. 16, 1916. The washings of B. C., age 3, taken during the acute stage of poliomyelitis, did not neutralize the filtrate. Immune serum had not been given.

While the number of observations is too small to draw definite conclusions, it is obvious that the secretions of apparently normal persons vary in the so-called neutralizing power. Of the two patients with poliomyelitis whose secretions inhibited action of the filtrate, one had received immune serum, while the washings were taken from the other on the 15th day, or at a time when immunity principles are known to be present in the blood.⁷ The third child with

7. Flexner, S., and Amoss, H. L., *J. Exp. Med.*, 1917, xxv, 499.

poliomyelitis yielded washings without neutralizing effect; but they were taken earlier (4th day) in the course of the infection and at a time when the immunity bodies were probably not yet abundantly present. It is possible that some relation exists between the presence of definite immunity principles in the circulating blood and the power of the nasal washings to neutralize the virus.

In each series of experiments the potency of the virus was established by control experiments, and subsequently all the monkeys not showing symptoms were tested for immunity by appropriate injections of the virus and were all found to have been susceptible to infection. Hence the lack of response was not caused by an immunity of the animals employed. The secretions of three persons out of six examined varied in their power to neutralize 0.2 cc. of the virus filtrate at different times under nearly identical conditions, yet the only known clinical differences consisted in the presence of a rhinitis which appears to remove the inactivating power of the secretions.

FLUCTUATIONS OF THE INACTIVATING POWER IN ABNORMAL NASAL CONDITIONS

In April, 1916, the nasal secretions of E. S. S. neutralized 0.2 cc. of the virus filtrate, but 3 months later, during an attack of acute rhinitis, they did not. The washings from C. A. R. twice neutralized the same amount of virus at different times. Later, immediately following an acute rhinitis, no neutralizing power was observed, but the neutralizing power returned in 4 days.

The washings from H. E. G., taken when rhinoscopy revealed acute congestion of the nasal mucosa, did not possess neutralizing power, but 5 weeks later when the nasal condition had improved, the washings showed the inactivating power. Six weeks after the second test when there were no subjective symptoms of rhinitis, the washings failed to neu-

tralize the virus. Finally, it will be recalled (Table III) that out of eight samples of nasal washings taken from apparently normal adults, only the two which were taken from subjects in which rhinoscopy revealed an acute rhinitis failed to inactivate the virus. H. E. G. is included in this list.

EFFECT OF FRACTIONAL STERILIZATION AND FILTRATION ON INACTIVATION

The experiments recorded indicate that the washings sterilized fractionally or passed through Berkefeld filters inactivate or neutralize virus mixed with them in the form of a filtrate of a suspension of the spinal cord of a poliomyelitic monkey. There can, therefore, be no doubt that the procedures do not themselves remove the neutralizing substances. Tests were then made to determine the comparative or quantitative effects of the procedures.

The quantity of filtrate employed for inoculation in this series of experiments was 0.4 cc., or more than four minimum lethal doses. The rinsings of the nasopharynx were made with redistilled water and they were reduced to a uniform volume of 15 cc. by concentration *in vacuo*. The fractional sterilization was carried out at 60°C. on 3 successive days. The washings and virus were left in contact 24 hours before the inoculations were made, in some instances at 37°C., in others at 4°C. The injections were intracerebral into *rhesus* monkeys under ether anesthesia. The results are given in Tables V and VI, and the comparison in Table VII.

The results lack absolute consistency. Considering the quantity of virus employed, the neutralizing action becomes more impressive. The variations in specimens from the same individual cannot now be accounted for. The existence of acute rhinitis, however, appears to diminish neutralizing power. Assuming that the process of neutralization is

brought about by definite chemical bodies, they would seem to be thermolabile, since the neutralizing action of filtrates is definitely more pronounced than that of the heated specimens. Contact at 4°C. appears less effective in bringing about the neutralization than at 37°C. The prolongation of the incubation period noted in two instances is probably associated with partial but insufficient neutralization to reduce the virus below the minimum lethal dose.

INFLUENCE OF HEAT

The results given above suggest that the inactivating influence is weakened or destroyed by heat. The following experiment gives more definite information concerning this fact.

Washings were taken on Nov. 16, 1916, from W.T., whose nasal secretions had on several occasions proved neutralizing. 60 cc. of sterile distilled water were used and the washings passed through a Berkefeld N candle.

To 10 cc. of washings filtrate were added 2.5 cc. of active virus filtrate and the mixture was incubated at 37°C. for 24 hours. 1 cc. of the mixture, representing 0.2 cc. of the virus filtrate, was injected intracerebrally, under ether anesthesia, into a *Macacus rhesus*. The monkey remained well.

35 cc. of the washings filtrate were reduced quickly *in vacuo* at a temperature between 60° and 70°C. to a volume of 5 cc. 1.25 cc. of active virus filtrate were added and the mixture was incubated at 37°C. for 24 hours. 1 cc. of the mixture, representing 0.2 cc. of virus filtrate, was injected intracerebrally, under ether anesthesia, into a *Macacus rhesus*. The monkey was completely paralyzed on the 7th day and died on the 8th day. Typical lesions of poliomyelitis were present.

The neutralizing substance is apparently rendered inactive by heating to 70°C., though this experiment does not exclude volatility as the reason for the disappearance of this substance. Other experiments, however, in which the concentrations were carried out *in vacuo* at 60°C. indicate that the neutralizing substances are not volatile.

TABLE V.

Neutralizing Power of Nasal Washings Heated to 60°C. for 1 Hour

<i>Mon- key</i>	<i>Dose of virus filtrate</i>	<i>Fractionally sterilized nasal washings from</i>	<i>Temper- ature at which virus plus nasal washings were incu- bated for 24 hrs.</i>	<i>Result</i>
	<i>cc.</i>		<i>°C.</i>	
A	0.4	W. T., normal adult	37	Died in 33 days
B	0.4	" " "	4	" " 31 "
C	0.4	G. H., " "	37	Remained well
D	0.4	" " "	4	Died in 11 days
E	0.4	C. A. R. (acute rhinitis)	37	" " 8 "
F	0.4	" " "	4	" " 8 "
G	0.4	Control (distilled water)	37	" " 14 "

TABLE VI.

*Neutralizing Power of Nasal Washings Passed through
a Berkefeld Filter*

<i>Mon- key</i>	<i>Dose of filtrate of virus</i>	<i>Berkefeld filtered nasal washings from</i>	<i>Temper- ature at which virus plus nasal washings were incu- bated for 24 hrs.</i>	<i>Result</i>
	<i>cc.</i>		<i>°C.</i>	
H	0.4	W. T. normal adult	37	Remained well
I	0.4	" " "	4	Died in 16 days
J	0.4	G. H., " "	37	" " 19 "
K	0.4	" " "	4	Remained well
L	0.4	C. A. R. (4 days after acute rhinitis)	37	" "
M	0.4	C. A. R. (4 days after acute rhinitis)	4	" "

TABLE VII.

Effect of Berkefeld Filtration and Heat on the Neutralizing Power of Nasal Washings

Nasal washings from	Condition of person from whom washings were obtained	Result of neutralizing test against 0.4 cc. of Berkefeld filtrate of virus			
		Filtered (Berkefeld) washings plus virus allowed to remain for 24 hrs.		Fractionally sterilized washings (60 °C. on 3 days) plus virus allowed to remain for 24 hrs.	
		37°C.	4°C.	37°C.	4°C.
W. T., adult	Normal	+	—	±†	±†
C. A. R., adult	Acute rhinitis	+	+	—	—
“ “	4 days after acute rhinitis	—	+	+	—
G. H., “	Normal	—	+	—	—
Control (distilled water)					

*The sign + indicates neutralization; ±, marked prolongation of the incubation period preceding paralysis.

†Incubation period greatly prolonged. Monkeys developed no symptoms until 33 and 31 days, respectively, after inoculation.

DISCUSSION

The power of the secretions of the nasopharynx of certain but not all individuals to bring about the inactivation or neutralization of the active virus of poliomyelitis has been demonstrated. The term active is employed to indicate that the samples of virus were obtained from strains adapted to the monkey, and could be relied upon to cause infection in the doses employed, almost without exception.

The inactivating property of the secretions mentioned is the more surprising in view of the resistance displayed by the poliomyelitic virus to such chemical antiseptics as glycerol and phenol.

In their manner of action, the neutralizing substances resemble more the specific immunity bodies contained within the blood serum of persons and monkeys who have suffered an attack of poliomyelitis. Like them, they appear to be

thermolabile. And yet the experiments here recorded do not actually identify the two classes of substances.

It is known that the blood serum of certain adults who apparently have never suffered from poliomyelitis is capable of neutralizing⁸ the filtered poliomyelitic virus.⁹ But in the few instances in which this property has been discovered, the adults yielding the serum had been in contact with acute cases of poliomyelitis, and artificial immunization cannot be excluded.

On the other hand, it seems not improbable that the inactivating or neutralizing power of the nasal secretions may play a part in protection against poliomyelitic infection, and even may represent an external system of defense against invasion of the virus by way of the nasopharyngeal mucosa.

If this view is supported by further studies, we should find that the secretions of children are less frequently neutralizing than those of adults, although many tests will be necessary to establish this distinction. In that case, we may find that the secretions of persons attacked by poliomyelitis at the period of onset of the disease lack neutralizing power, although later, when the immunization reactions have been aroused, inactivation may result, as has been shown to happen in particular instances in our series.

It appears, however, that the power of a given secretion to inactivate or neutralize the virus is not wholly a fixed one. Fluctuations in the property have been detected and described. Common and slight inflammatory conditions, *e.g.*, as in acute and even chronic rhinitis, apparently tend to remove or diminish the neutralizing power of the secretions. If this observation should be supported by further experiment, knowledge concerning one of the conditions favoring persistent contamination of the nasopharynx with the virus may be obtained. It does not follow, however, that

8. Flexner and Lewis, *J. Am. Med. Assn.*, 1910, liv, 1780.

9. Peabody, F. W., Draper, G., and Dochez, A. R., *A Clinical Study of Acute Poliomyelitis*, Monograph of The Rockefeller Institute for Medical Research, No. 4, 1912.

this contamination need necessarily lead to infection, for the accomplishment of which disturbance of still other defensive mechanisms may be necessary. However, the production of healthy carriers of the poliomyelitic virus may rest upon the power or lack of power in the secretions to inactivate the virus. Should this be the case, then of many persons exposed only a fraction would become carriers, because the greater part would possess secretions capable of neutralizing and hence destroying the virus.

The variation in inactivating power does not depend alone upon inflammatory changes. Irregularities have been noted which cannot now be explained. They may be merely apparent and depend upon the experimental method to which we are at present limited. Inoculation experiments in single series are not wholly trustworthy. Filtration through porcelain is also open to errors of experiment, since the blocking of the porous spaces may easily exclude essential constituents of the washings. Fractional sterilization is also not a wholly reliable means of preventing bacterial development and yet of retaining unimpaired labile organic constituents. In view of all this, some degree of irregularity is to be looked for.

If this property of the secretions to inactivate or neutralize the virus of poliomyelitis is established, comparative tests should be made on large groups of persons at different seasons of the year in order to determine whether it bears any relation to the seasonal prevalence of poliomyelitis. We are engaged now in collecting observations covering this point; but reference to the tables will show that most of the tests were made during the spring, summer, and autumn. Moreover, they embraced few children of the most susceptible ages.

SUMMARY

1. The results of 56 experiments have shown that washings of the nasal and pharyngeal mucosae possess definite

power to inactivate or neutralize the active virus of poliomyelitis.

2. This power is not absolutely fixed, but is subject to fluctuation in a given person. Apparently inflammatory conditions of the upper air passages tend to remove or diminish the power of neutralization. But irregularities have been noted, even in the absence of these conditions.

3. Too few tests have been made thus far to ascertain whether adults and children differ with respect to the existence of this neutralizing property in the nasal secretions. While the inactivating property was absent from the secretions of one child during the first days of poliomyelitis, it was present in another to whom immune serum was administered, and in still another on the 15th day of illness when convalescence was established.

4. The neutralizing substance is water-soluble and appears not to be inorganic; it appears to be more or less thermolabile, and its action does not depend upon the presence of mucin as such.

5. It is suggested that the production of healthy carriers through contamination with the virus of poliomyelitis may be determined by the presence or absence of this inactivating or neutralizing property in the secretions. Whether this effect operates to prevent actual invasion of the virus and production of infection can only be conjectured. Probably the property is merely accessory and not the essential element on which defense against infection rests. It is more probable that other factors exist which help to determine the issue of the delicate adjustment between contamination and infection.

CARRIAGE OF THE VIRUS OF POLIOMYELITIS, WITH SUBSEQUENT DEVELOPMENT OF THE INFECTION*

By EDWARD TAYLOR, M.D.

AND

HAROLD L. AMOSS, M.D.

*(From the Research Laboratory† of the Vermont State
Board of Health, Burlington.)*

THE solution of the problem of the mode of infection in poliomyelitis has been attempted in various ways, with results which have led to the conclusion that the microbic cause is conveyed from one individual to another by personal contact. This belief is based upon clinical observation and experiment. Wickman first brought clinical proof, since supported by many independent observations, of the correctness of this generalization; and Flexner and Lewis, and later Kling and Pettersson, provided the experimental demonstration of its adequacy.

However, a considerable number of physicians and others still refuse to accept this explanation. They hold that the mode of infection remains undiscovered, or they account for it through some variety of insect transmission, also undetected. In recognition of the skepticism still prevailing, we have been led to describe in detail the experimental demonstrations of the carriage by healthy persons of the virus of poliomyelitis, to which may now be added our own successful inoculations. Our results include the demonstration, recorded for the first time, that a proved carrier of the virus

*Reprinted from *The Journal of Experimental Medicine*, November 1, 1917, Vol. XXVI, No. 5, pp. 745-754.

†Maintained by a special fund privately donated.

may come down with acute poliomyelitis. This observation should serve to strengthen the position of those who accept as established the personal communication of the microbic cause, or virus, of the disease.

PREVIOUS REPORTS

Wickman's¹ clinical studies may be said to have disseminated the view of the personal factor in the communication of the virus of poliomyelitis. He emphasized the occurrence and epidemiological importance of the non-paralytic or abortive cases, the first description of which is usually credited to him, and of healthy intermediaries, or bacillary carriers, who function as purveyors of the microbic agent. His study constituted a great step forward; but the first person to allude to non-paralytic cases of epidemic poliomyelitis is Caverly,² who records the occurrence of 6 cases among the total of 132 cases on which he based his report describing the Rutland epidemic of 1894.

Soon after Landsteiner and Popper's³ experimental transmission of poliomyelitis, Flexner and Lewis⁴ detected the virus in the nasopharyngeal mucous membrane of infected monkeys. This observation, soon confirmed by several independent bacteriologists, was followed by a study made by Kling, Pettersson, and Wernstedt⁵ who injected into monkeys buccal washings from so-called abortive cases and from healthy contacts. Their results were inconclusive, as the clinical condition produced was not typical of poliomyelitis, and the pathological changes described as present in the spinal cord were not characteristic of the disease. They explained the discrepancy by the supposition that the virus present in the abortive cases and healthy carriers was relatively avirulent. This view is repeated in their recent report⁶ in which they describe an instance of healthy carriage of the highly active virus inducing paralysis and characteristic lesions. The first demonstration of the typical virus in the nasopharyngeal washings of healthy persons was, however, made by Flexner, Clark, and Fraser,⁷ whose report follows in detail.

E. A., female, age 4 years and 4 months. The patient had been ill from Oct. 12 to 17, 1912. On the latter date she was admitted to the Hospital of The Rockefeller Institute for Medical Research, suffering from severe paralytic poliomyelitis. She subsequently improved and was discharged, Oct. 28. The mother and father of the child were subjected

to a nasopharyngeal irrigation with normal saline solution; about 150 cc. of washings were obtained. The fluid was shaken and passed through a Berkefeld filter; of the filtrate, 1.5 cc. were injected the same day into the sheath of each sciatic nerve and 140 cc. into the peritoneal cavity of a *Macacus cynomolgus* (Monkey A). Recovery from the anesthesia was prompt and the animal remained well until Nov. 11, when it was noted to be excited and to drag the right leg; the left leg was weak. Nov. 12. Right leg flaccid. A lumbar puncture yielded 2.5 cc. of fluid containing excess of white corpuscles. Nov. 13. The condition was unchanged; the animal was etherized. The organs generally were normal in appearance; the spinal cord was edematous. Microscopic examination of sections of the spinal cord, medulla, and interstitial ganglia revealed the characteristic lesions of poliomyelitis. The blood vessels and ground substance showed infiltrations with mononuclear cells; the motor nerve cells were degenerated and invaded by phagocytes.

Dec. 3. An emulsion of the glycerolated spinal cord and medulla was injected into each sciatic nerve and the peritoneal cavity of a *Macacus cynomolgus* (Monkey B) and a *Macacus rhesus* (Monkey C). Dec. 9. The *rhesus* monkey was noted to be excited. Dec. 10. Lumbar puncture yielded 3 cc. of turbid fluid containing excess of white cells. By Dec. 13, the legs were partially paralyzed; the animal was etherized. Microscopic sections of the spinal cord, medulla, and intervertebral ganglia showed typical infiltrative and degenerative lesions of poliomyelitis. The *cynomolgus* monkey became excited on Dec. 10, and on the 19th paralysis of the legs appeared. By Dec. 21 the arms and back were weak, and the paralysis was extending. Dec. 23. The animal was etherized. The general viscera appeared normal, but the spinal cord was both edematous and congested. The microscopic sections of the cord, medulla, and intervertebral ganglia showed typical infiltrative and degenerative lesions attended by neurophagocytosis. Subsequently the glycerolated specimens of the nervous organs of Monkeys B and C were used for inoculating still other monkeys, in which typical paralysis was induced.

The conclusion drawn by the authors from this demonstrative experiment was to the effect that the parents of 2 liters from healthy persons in contact with cases of acute E. A., neither of whom showed any symptoms of illness and who evidently were not suffering from poliomyelitis, harbored the virus of the disease in the nasopharynx. Hence the

existence of the healthy carrier was thus established experimentally.

The next demonstrative experiment was supplied by Kling and Pettersson⁶ who, in referring to their earlier failure to produce clinically and anatomically typical poliomyelitis with nasopharyngeal washings, attribute the failure to the injection of insufficient amounts of virus into the monkeys. They repeated the tests, using washings concentrated *in vacuo* with the Faust-Heim apparatus.

They started out by determining the heat lability of the active virus, and ascertained that a liter of fluid carrying an effective dose could be evaporated at temperatures ranging from 35 to 38°C. to 200 cc. without losing its potency. They now obtained nasopharyngeal washings in amounts of 1 to poliomyelitis. In one instance in which the washings were taken from the healthy members of a family in which one member had recently died of acute poliomyelitis, the inoculation resulted successfully.

The patient was a male, age 41 years. The illness began on Sept. 10, the legs becoming paralyzed 2 days later. Death took place on the 4th day of illness from respiratory failure. The surviving members of the family consisted of the wife and three children, ranging from 10 to 14 years, all remaining well. One day after the death of the father in a hospital, nasal washings were taken in distilled water from the surviving members of the family. The combined washings, amounting to 1 liter, were evaporated *in vacuo* to 75 cc., sodium chloride was added, and the mixture was filtered first through paper and then through a Berkefeld candle.

Sept. 20. 0.5 cc. of the filtrate was injected intracerebrally and 20 cc. were introduced into the peritoneal cavity of a *Macacus sinicus*. Oct. 2. The right leg and on the next day both legs and back were paralyzed, and death resulted. The microscopic sections of the spinal cord showed moderate perivascular and diffuse infiltration of the nervous tissue with mononuclear cells and neurophagocytosis. Oct. 3. A second *Macacus sinicus* was inoculated intracerebrally and intraperitoneally with an emulsion of the spinal cord of the first animal. On Oct. 13 the right leg and on the next day the left leg were paralyzed. Oct. 15. The animal was killed. Sections of the spinal cord showed typical infiltrative and degenerative lesions of poliomyelitis.

There can be no doubt, therefore, that in this family one or more healthy carriers of the active virus of poliomyelitis

existed. That the result was not due entirely to the employment of concentrated washings is indicated by the failure to detect the virus in the washings obtained from the healthy associates of two other cases of acute poliomyelitis.

OBSERVATIONS

In the two successful instances just reviewed, mixed washings were employed for inoculation. It is, therefore, impossible to state whether one or more of the healthy contacts of the cases of poliomyelitis were carriers. In the instance which we shall report the individuals were irrigated separately. The final result proved that more than one virus carrier was present, and it was demonstrated that such a healthy carrier may develop poliomyelitis. We may therefore regard the chain of the mode of infection as now having been completed for the first time. The separate links may be defined as follows:

Case of acute poliomyelitis → contact carrier → second case.

A still further analysis would determine that through the contact carrier other carriers occur, among which a certain number of additional cases arise.

Poliomyelitis occurred in epidemic form in Washington County, Vermont, in the summer of 1917. From June 1 until September 1, 79 cases were recognized among the population of 45,000.

Carey P., male, age 16 years. The patient lived in the village of Waitsfield, 18 miles from Montpelier, where cases of poliomyelitis existed. No case of the disease had been discovered in Waitsfield. On June 2, 1917, he attended a ball game at Northfield where there were no cases, and returning home stopped in Montpelier for supper. Probably in the assembly at Northfield persons from the infected district were present. Until June 12 there were no symptoms of illness; on that day there was complaint of headache and pain in the back and legs. The patient vomited once. June 13. First seen by a physician who observed that the patient

had fever, and treated him for a gastrointestinal upset. June 16. Extensive paralysis involving both legs, right triceps, intercostals, pectorals, and diaphragm. Lumbar puncture yielded clear fluid under pressure, containing 400 white cells per cmm. and excess of globulin. Death occurred on this date.

The family consisted of the father, age 59 years, mother, age 42, sister, Hazel, age 13, two brothers, Everett, age 10, and Dwight, age 7. The two younger brothers slept in the same bed, and in the same room with the elder brother, Carey.

June 16. Everett and Hazel were given nasopharyngeal irrigation with distilled water, 60 cc. being obtained from the former and 100 cc. from the latter. Ten per cent of ether was added to each, and the fluids were sent at once to the laboratory. One of us had previously determined that ether inhibits bacterial development without injuring the poliomyelitic virus. The washings were treated separately as follows: Glass beads were added and they were shaken mechanically for $2\frac{1}{2}$ hours. They were then centrifuged at high speed for $2\frac{1}{2}$ minutes, and the supernatant fluid was passed through a Berkefeld N candle and concentrated *in vacuo* by the method already described by us⁸ at 35°C. to 2 cc. The entire concentrate was injected intracerebrally into two *Macacus rhesus* monkeys (Monkey A (Everett) and Monkey B. (Hazel)). The time elapsing between the collection and the injection of the washings was less than 6 hours.

We return briefly to the history of the two children. Everett had not been away from the village and was in usual health until June 13, the day after Carey fell ill. He also felt indisposed, showed a temperature of 102° F. and suffered from diarrhoea, but did not vomit. However, he recovered quickly and subsequently on minute examination has shown no muscular weakness or abnormality of reflexes.

Hazel had not been away from Waitsfield. She had been

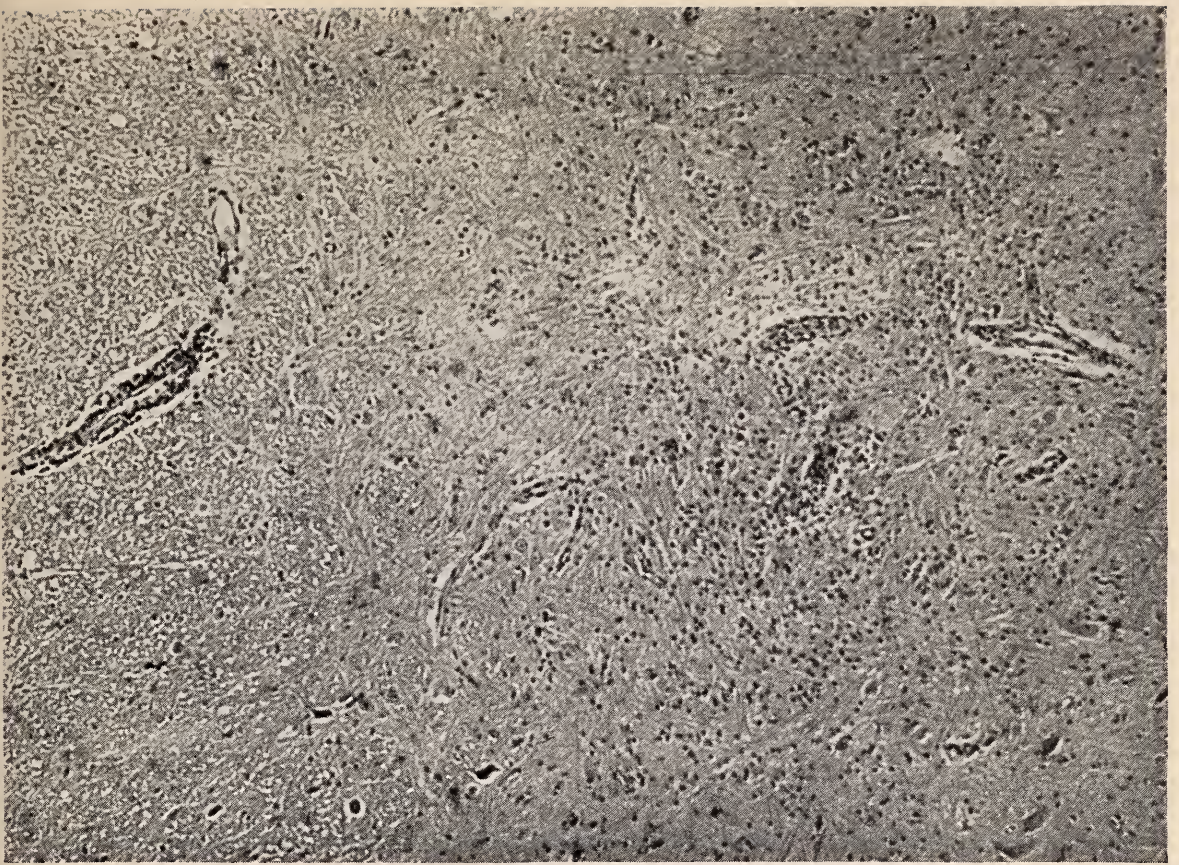
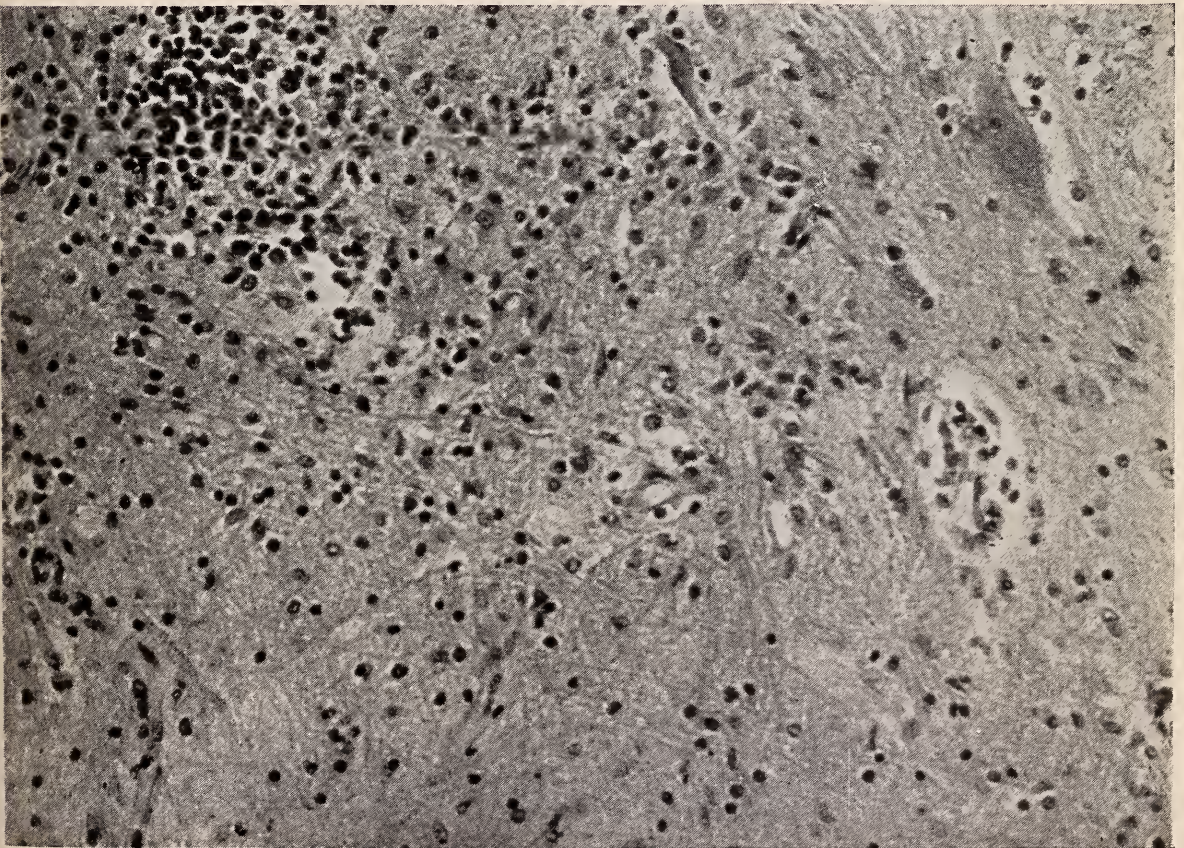


FIG. 1. Spinal cord of Monkey B, showing perivascular infiltration and neurophagocytosis. $\times 90$.

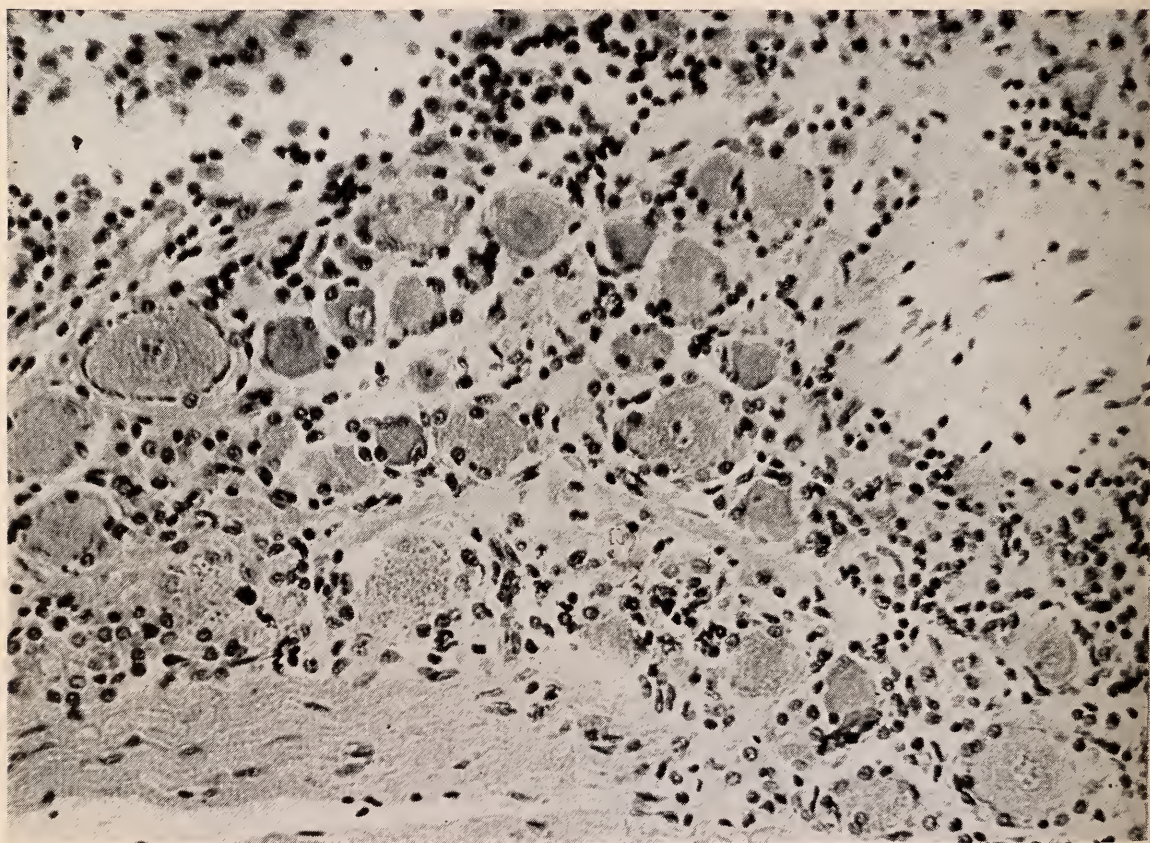


(Taylor and Amoss: Carriage of the virus of poliomyelitis.)

FIG. 2. Medulla of Monkey B, showing diffuse mononuclear infiltration, nerve cell degeneration, and neurophagocytosis. $\times 230$.



FIG. 3. Intervertebral ganglion of Monkey B, showing infiltrative changes and nerve cell invasion. $\times 120$.



(Taylor and Amoss: Carriage of the virus of poliomyelitis.)

FIG. 4. Intervertebral ganglion of Monkey B, showing mononuclear infiltration, nerve cell degeneration, and neurophagocytosis. $\times 240$.

entirely well at the time the washings were taken and remained well until June 21, at which time she complained of headache. She showed a temperature of 102° F. On June 22 her reflexes were exaggerated and stiffness of the back was present, but no muscular weakness was detected. Lumbar puncture was unsuccessful. The symptoms subsided gradually, but reexamination made on July 22 revealed partial paralysis of the left deltoid, right anterior tibial, and abdominal muscles. She had, therefore, suffered a mild attack of poliomyelitis.

Monkey A.—June 16, 1917. Inoculated. Remained well until June 29, when it was excitable, emitted staccato cries, and showed ruffled hair. The animal was noted to be clumsy in movement and unable to jump. June 30. Both legs were weak. July 4. The right leg was paralyzed and flaccid; the left leg and back were weak. The paralysis of the left leg and back, but not of the right leg, disappeared; the latter remained and contracture gradually set in. At the present time (Sept. 1) the contraction of the right leg is so marked that in moving about the animal does not touch the limb to the floor. On Aug. 8 blood was withdrawn for a neutralization test and at the same time an intracerebral inoculation was made with a large dose of virus proved active in another monkey; the result was negative. The animal, as is usually the case, having recovered from a recent infection, was resistant to reinoculation.

Monkey B.—June 17, 1917, 3 a.m. Injected intracerebrally with 1.5 cc. of the concentrated washings. Recovery from the anesthesia was immediate, and the first symptoms, consisting of excitability, ruffled hair, staccato cries, and partial paralysis of the right leg, were observed. June 26. The paralysis being stationary, the animal was etherized. The organs appeared normal to the naked eye. Microscopic sections revealed, however, marked typical lesions of poliomyelitis. They affected the spinal cord (Fig. 1), medulla (Fig. 2), and intervertebral ganglia (Figs. 3 and 4), and consisted of typical infiltration with mononuclear cells and nerve cell degeneration with phagocytosis.

Monkey C.—June 26, 1917. Injected intracerebrally under ether anesthesia with 2.5 cc. of a 20 per cent emulsion of spinal cord and medulla of Monkey B. July 7. The first symptoms were noted, consisting of ruffled hair and inclina-

tion of head to the left. July 8. The animal was ataxic and protected the right leg. July 9. Unable to jump; legs and back weak. July 10. Paralysis progressing. July 15. Etherized. The spinal cord showed typical focal lesions of poliomyelitis in which cicatrization was beginning.

These experiments leave no doubt that the washings, both from Everett and from Hazel, contained the virus of poliomyelitis. The instance of Hazel is of particular importance since in her case the virus was detected in washings taken 5 days before the first symptoms of what proved subsequently to be a mild attack of poliomyelitis set in. In other words, she was carrying the virus in her nasopharynx several days in advance of the appearance of any signs of illness. She constitutes, therefore, an example of a carrier of the virus developing poliomyelitis—the first one in which the demonstration has been proved experimentally.

The interpretation in the case of Everett is not so simple. When the virus was detected in his nasopharynx he had passed through a slight attack of illness, at about the same time with, and of about the same character as that of his brother Carey who died, but unattended by paralysis. The presumption is that Everett suffered from a non-paralytic or abortive attack of poliomyelitis. The detection of the virus in his case proves him not to have been a healthy, but a recovered carrier of the microbic cause of the disease.

The two children having been shown to be virus carriers, their nasopharyngeal secretions were tested by the method of Amoss and Taylor,³ to determine whether they would neutralize an active poliomyelitic virus.

July 23, 1917. Washings with sterile water were taken from the children, and fractionally sterilized and mixed. To 15 cc. of the mixture were added 3.75 cc. of a Berkefeld filtrate of a 5 per cent stock glycerolated poliomyelitic spinal cord. After shaking, the combined fluids were permitted to remain at 37°C. for 24 hours. 1 cc. of the fluid was injected intracerebrally into a *Macacus rhesus*. No symptoms ap-

peared until Aug. 4, when excitability, ataxia, paralysis of the right arm, and weakness of the back were noted. Aug. 8. Animal prostrate. Aug. 10. Died. The microscopic lesions were typical of poliomyelitis.

The mixed nasal washings failed, in this experiment, to neutralize the virus.

The youngest child, Dwight, age 7 years, was refractory and no washings were obtained from him on June 16 when they were taken from the other children. On June 18 he complained of being unwell. The symptoms were severe headache, stiffness of neck, exaggerated reflexes, but no diarrhoea. Lumbar puncture yielded a fluid containing 500 white cells per cmm. and an excess of globulin. Immune poliomyelitic serum from recovered cases of the disease was administered intraspinally, intravenously, and subcutaneously: 24 cc. were given intraspinally, 30 cc. intravenously, and 39 cc. subcutaneously. Recovery was prompt, with a slight paralysis of the right anterior tibial muscle. Nasopharyngeal washings were, however, obtained on September 4, which after filtration and concentration were inoculated into a *Macacus rhesus* (Monkey D). The monkey remained well.

DISCUSSION

This series of cases of poliomyelitis in one family, with the circumstances surrounding their origin, forms an instructive illustration of the mode of infection of the disease as brought out by the clinical and experimental study.

In the first place, one child only—the eldest boy, Carey—was exposed in a locality in which poliomyelitis was epidemic. The exposure took place on June 2. Immediately afterwards he returned home, to a village in which no previous case of the disease had occurred, and mingled freely with his younger brothers and sister. The contacts may be considered to have been intimate in that the three male children slept in the same room, two of them in the same bed.

The incubation period in Carey's case was 9 or 10 days, as he was taken ill on June 12. His brother Everett, 6 years younger, developed symptoms 1 day later and passed through what was probably a non-paralytic attack of poliomyelitis. He may be considered as having been infected by Carey some time during the incubation period, and to have exhibited a shorter incubation than his brother. The youngest brother, Dwight, was also freely exposed to both older brothers and exhibited symptoms passing into those indicative of poliomyelitis 5 or 6 days later than his brothers. Finally, Hazel, the sister, in age between the two older brothers and possibly less freely exposed, developed symptoms and muscular weakness last of all and about 10 days after the eldest brother. The incubation periods of the cases, therefore, probably were 10 days or less, and the order of the attacks was such as to indicate successive infection and not a common one.

The second feature worthy of emphasis is the detection in this one family of two carriers of the poliomyelitic virus by the inoculation test. One (Everett) was discovered to be a carrier probably following a non-paralytic attack. In the instance of Hazel there is no doubt, first that she was discovered to be a healthy carrier, and second that she developed typical poliomyelitis during the period of carriage. Incidentally the nasopharyngeal secretions of Hazel and Everett failed to neutralize the poliomyelitic virus.

If the view that the mode of infection in epidemic poliomyelitis is by way of the nasopharyngeal mucous membrane and is brought about or greatly facilitated through the operation of healthy carriers of the virus is accepted, we may well consider whether in the final analysis every case of the disease does not develop from a carrier. At first this may seem startling, and yet it merely means that after contamination of the nasopharynx with the virus, an intervening period exists during which persistence, multiplication, and

invasion of the virus take place. In not all contaminated persons does this process become complete; in some the virus may merely persist for a time, in others it may multiply in the nasopharynx (these constitute the healthy carriers of greater or less endurance), while in the exceptional few invasion also occurs. In the last class symptoms arise, and these individuals become cases of poliomyelitis.

SUMMARY

A family group containing four children of whom all showed in varying degree symptoms of poliomyelitis is described. The source of infection and periods of incubation have been followed. Two of the children were proven by inoculation tests to carry the virus of poliomyelitis in the nasopharynx. Of these, one was detected to be a carrier after recovering from a non-paralytic attack of the disease, and the other was discovered to be a carrier about 5 days before the initial symptoms, attended later by paralysis appeared. The original case from which the three others took origin was fatal; the youngest child, after quite a severe onset, was treated with immune serum, and made a prompt and almost perfect recovery. The nasopharyngeal secretions of two of the cases, taken 1 month after the attack, proved incapable of neutralizing an active poliomyelitic virus.

The proposition is presented that every case of poliomyelitis develops from a carrier of the microbic cause, or virus, of poliomyelitis.

1. Wickman, I., Beiträge zur Kenntnis der Heine-Medinschen Krankheit. Berlin, 1907.

2. In view of the importance which the non-paralytic cases have assumed in the epidemiology of poliomyelitis it is pertinent to quote Caverly, who states that paralysis occurred in 119 cases, 7 cases died before paralysis was detected, "and the remaining 6 had no paralysis, but all had a group of symptoms very common in the initial stage in those which were paralyzed, such as headache, fever, convulsions, or nausea, one or all" (*J. Am. Med. Assn.*, 1896, xxvi, 1).

3. Landsteiner, K., and Popper, E., *Z. Immunitätsforsch., Orig.*, 1909, ii, 377.

4. Flexner, S., and Lewis, P. A., *J. Am. Med. Assn.*, 1910, liv, 1140.

5. Kling, C., Pettersson, A., and Wernstedt, W., *Communications Inst. méd. État a Stockholm*, 1912, iii, 5.

6. Kling, C., and Pettersson, A., *Deutsch. med. Woch.*, 1914, xl, 320.

7. Flexner, S., Clark, P. F., and Fraser, F. R., *J. Am. Med. Assn.*, 1913, lx, 201.

8. Amoss, H. L., and Taylor, E., *J. Exp. Med.*, 1917, xxv, 507.

THE TREATMENT OF ACUTE POLIOMYELITIS†

PRELIMINARY NOTE ON USE OF HYPERTONIC SALT SOLUTION AND CONVALESCENT HUMAN SERUM*

WILLIAM LLOYD AYCOCK, M.D.

AND

HAROLD L. AMOSS, M.D.

THE results in general of the serum treatment of acute poliomyelitis have not been sufficiently consistent to warrant a definite statement as to its value. In the absence of hyperimmune serum, recourse must be had to human convalescent serum, which is at best weak in its antibody content. In a small series,¹ the administration of large doses of convalescent serum a few hours after onset yielded distinctly encouraging results, but it is agreed that the problem has not been solved. Such treatment is beset with obstacles apparently insurmountable, one of the greatest of which is the inaccessibility of the site of injury in poliomyelitic infection.

Since the flow of fluid within the nervous tissue itself is probably from the capillaries along the pericapillary, perineuronal and perivascular spaces toward the subarachnoid space, serum injected merely into the subarachnoid sac cannot be expected to reach the inflammatory focus lying deep in the gray matter of the cord. It is likewise true that the walls of the capillaries of the nervous tissue constitute an effective barrier against the passage of serum from the

†Copyright, 1923, Am. Med. Assn. Reprinted by permission from J. of A. M. A., Aug. 11, 1923, Vol. 81.

* From the Research Laboratory (maintained by a special fund privately donated), Burlington, Vt., and the Biological Division of the Medical Clinic, Johns Hopkins Hospital and University.

1. Amoss, H. L., and Chesney, A. M.: J. Exper. Med. 25: 581 (April) 1917.

circulation to the perivascular system.² Weed and his co-workers³ have shown that the intravenous injection of hypertonic solutions in normal animals causes a reduction in the volume of the brain and spinal cord with a marked lowering of the cerebrospinal fluid pressure and an aspiration of fluid from the subarachnoid space into the perivascular "lymph" spaces of the brain and cord.

In our experiments,⁴ similar results have been obtained in the edematous cords of monkeys in the acute stage of poliomyelitis, and have led to the employment of the method in the treatment of the experimental disease. Poliomyelitic monkeys given convalescent human serum intraspinally and intravenously, and hypertonic salt solution intravenously, exhibited marked improvement as compared to controls. By the shrinking of the central nervous tissues after intravenous injection of hypertonic salt solution, more space is available in the subarachnoid space for the reception of large amounts of serum; this is especially advantageous, since the convalescent serum is at best weak in antibody content, and correspondingly larger amounts must be injected. The beneficent effect of the change in tonicity of the blood, with the marked decrease in the volume of the brain and spinal cord, is to reduce the inflammatory edema of the cord. However, it seems that edema cannot be explained entirely on the basis of osmosis. Our experiments have not progressed sufficiently to warrant any statement on this point.

The third and probably the main possibility of a beneficent effect of such a method lies in the fact that the intravenous injection of hypertonic solution brings about an aspiration of serum from the subarachnoid space into the

2. Mott, F. W.: *Lancet* 2: 79, 1910. Flexner, Simon: *The Local Specific Therapy of Infections*, J. A. M. A. 61: 447 (Aug. 16) 1913. Flexner, Simon, and Amoss, H. L.: *J. Exper. Med.* 25: 499 (April) 1917.

3 Weed, L. H.: *J. M. Res. N. S.* 26: 93, 1914. Weed, L. H., and Hughson, Walter: *Am. J. Physiol.* 58: 53, 101 (Nov.) 1921. Weed, L. H., and McKibben, P. S.: *Ibid.* 48: 531, 1919. Weed, L. H.: *Am. J. Anat.* 31: 191 (Jan.) 1923.

4. These will be reported in the *Bulletin of the Johns Hopkins Hospital*, December, 1923.

perivascular system, thus insuring a more intimate contact between the main lesions of poliomyelitis and the serum, which can be regarded as a distinct advantage in local specific therapy. There is one other possibility in connection with the employment of this method. It is possible to administer enough hypertonic solution, approximately 1 gm. of sodium chloride per kilogram of body weight, to cause cerebrospinal fluid pressure to fall from 80-120 mm. of water to —90 mm. of water without damage. This loss of fluid within the central nervous system is replaced afterward by an increased passage of fluid from the blood stream to the cerebrospinal fluid, normal pressure being reestablished within a few hours. On the hypothesis that this increased flow of fluid would facilitate the passage of serum from the circulation to the fluid spaces of the central nervous tissue, the intravenous injection of immune serum one or two hours after the injection of hypertonic solution is recommended.

REPORT OF CASE

Opportunity for the application of this method to cases in human beings has not yet been afforded except in one instance. It is realized that, without data from many cases, no conclusions can be drawn; yet, owing to the demand for details of the method a brief report of one case is given.

History.—G. E. I., a boy, aged 4 years, with normal development, and past history unimportant, entered Harriet Lane Home⁵ of the Johns Hopkins Hospital, Jan. 16, 1923, for treatment for acute abdominal pain and sudden loss of power to move the arms and legs, closely following a gastrointestinal upset. The onset occurred January 8, with repeated chills and fever, which endured for two days. On the fourth day, the symptoms disappeared, and the patient remained asymptomatic until the morning of January 15, when he told his mother that he could not “wiggle his toes.” The weakness of the legs noticed at that time gradually in-

5. The patient was under the care of Dr. W. J. Scott, from whose notes this abstract was taken.

creased until motion was lost. Tingling in the fingers appeared, and there was a rapid loss of motor function of the arms and trunk. On admission, the temperature was 100.4 F., the respirations, 40, and the pulse, 120. The patient was mentally clear and cooperative. The extremities were flaccid, with perhaps slight power in the right arm and leg. The patient was unable to move in bed. Speech was normal.

Examination.—The head, eyes, ears, sinuses, nose, throat and mouth were normal. There was no glandular enlargement; the chest, heart, lungs, abdomen, genitalia and joints were normal. The extremities were flaccid, as already described.

The tendon reflexes of the upper and lower extremities could not be obtained; the abdominal reflexes were hyperactive. There was no clonus, and no Babinski's sign on either side; Kernig's sign was negative. There was no abnormality of the cranial nerves, and no disturbances of sensation. Flexion of the neck and back produced pain.

Urine examination was negative. Blood examination revealed: red blood cells, 5,288,000 (cells normal); white blood cells, 19,500. Spinal fluid withdrawn at 7:30 p. m., January 16, was clear and under normal tension; there were 40 cells, mostly mononuclear, and the globulin test was negative; the Wassermann reaction was negative. An intracutaneous injection of 0.1 mg. of tuberculin gave a positive reaction. A roentgenogram of the chest showed no abnormality.

Course and Treatment.—At noon, January 17, it was noticed that the facial muscles on the right side, and intercostal muscles on both sides were weak. Both anterior and posterior deltoid muscles on the left side were completely paralyzed, and all the muscles of the right arm were weaker than normal. The right leg was now completely paralyzed.

It seemed entirely reasonable to suppose that this was a case of acute poliomyelitis of Landry's type, and the subsequent course substantiates this view.

On the basis of results obtained in the treatment of experimental poliomyelitis in monkeys, treatment with intravenous hypertonic solution and convalescent human serum was begun.

January 17, at 8 p. m., lumbar puncture was performed; 35 cc. of clear spinal fluid was removed, and human convalescent poliomyelitic serum allowed to flow in until equilibrium was established with a head of 4 inches. It was estimated that 20 c.c. of serum flowed into the subarachnoid space. While the needle and connections remained in place,

there was injected, intravenously under ether anesthesia, 25 c.c. of concentrated Ringer's solution, of which the sodium chloride content was 18 per cent. Within two minutes, the serum began to flow again into the spinal canal at a fairly rapid rate. After 20 c.c. of serum had been allowed to flow in, there was no apparent decrease in the rate of flow, and the needle was withdrawn. Since there was already obvious involvement of the respiratory center, the injections were made with dispatch, and no manometric readings were made.

During the night, the patient was very thirsty; a small amount of crushed ice was allowed, but water was withheld. By 1 o'clock that night, the rectal temperature reached 104.4. There were two short convulsions and convulsive movements of the back muscles. Fluids were given by mouth, and the temperature was reduced to 102. The patient was very restless.

January 18, at 10 a. m., lumbar puncture was again performed. The fluid obtained was turbid, with 18,000 cells per cubic millimeter, most of them polymorphonuclear. The temperature became normal at 7 p. m. The white blood cell count was 25,000. At 10 p. m., the diaphragm was weaker, and the abdominal muscles were being used more than usual.

January 19, at 9 a. m., the patient seemed better. Five per cent glucose solution by rectum was not retained. Fluids were given by mouth in small amounts. The patient could swallow, but seemed unable to cough. The chest was clear, and the respiratory rate was from 26 to 30 a minute.

January 20, respirations were improved, and the patient seemed brighter. He complained at times of pains in the legs.

January 21, the patient seemed very bright, and the facial weakness had almost disappeared.

January 22, he was breathing with less difficulty. There were four loose, involuntary stools.

January 23, there were no more involuntary stools, and the patient voided normally. He was hungry for the first time. Breathing was improved, but there was still a noticeable weakness of the diaphragm.

January 25, there was a return of the inability to move the fingers and toes.

January 27, there was improvement in the muscles of the shoulder.

January 28, the muscles of the arm were stronger.

January 30, the patient moved the fingers of both hands

for the first time, the right better than the left. Neither thumb functioned. The triceps muscle on the right functioned. The left arm was flaccid, but could be moved slightly with effort. There was slight power in the right biceps, and none in the left.

February 1, there was slight voluntary contraction of both adductors.

February 3, the diaphragm and intercostal muscles were definitely stronger.

February 4, there was a slight return of power in the left triceps muscle; the right was stronger.

February 10, there was definite improvement of the muscles previously mentioned.

February 23, there were movements in the right leg.

The patient continued to improve, and was discharged, March 29. Monthly observations have revealed a steady improvement and excellent restoration of function of the muscles of the upper extremities. The patient was seen last, June 14. At that time there was still some weakness of grip in the left hand, and slight weakness of the posterior deltoid on the right, but he was able to raise both arms above his head, to flex his thighs on his abdomen, turn over in bed and sit up. There was slight motion in the toes of the right foot—none in the left.

COMMENT AND SUMMARY

The story, taken as a whole, conforms well to the type of Landry's paralysis seen in epidemics, but unusual in sporadic cases of poliomyelitis. In such a case presenting first paralysis of the extremities rapidly extending and finally developing respiratory paralysis, the prognosis was very grave, and it was because of the apparent hopeless outlook that a method of treatment not yet fully developed and hitherto employed only in experimental poliomyelitis was applied.

Whether the intravenous injection of hypertonic salt solution stayed or slightly cleared the edema of the cord, or brought the convalescent human serum nearer the site of the inflammation, or only made it possible to inject more serum intraspinaly without danger of pressure, is not yet clear.

This case is described in order to present details of the method of the treatment, and no claim whatever is made that the patient was benefited by the injections. Apparently no harm was done by the procedure.

On account of the limited amount of serum on hand, the intravenous injection of 100 c.c. of convalescent human serum after the administration of hypertonic salt solution was omitted. This plan is recommended on the basis of experiments to be described in a forthcoming paper. In these experiments, it was found that, while no especial difficulty was encountered in initial injections of hypertonic solutions, daily repetition of the injection in poliomyelitic monkeys caused respiratory failure sometimes after comparatively small amounts of hypertonic solution had been injected. Hence, repeated injections cannot be recommended.

We are indebted to Dr. John Howland for permission to study and report this case.

EXPERIMENTS ON LOCAL SPECIFIC THERAPY IN POLIOMYELITIS*

THE UTILIZATION OF HYPERTONIC SOLUTIONS IN THE SERUM TREATMENT OF EXPERIMENTAL POLIOMYELITIS

By W. L. AYCOCK, M.D.

AND

H. L. AMOSS, M.D.

*(From the Research Laboratory† of the Vermont State
Board of Health, Burlington, and the Biological Divi-
sion of the Medical Clinic, Johns Hopkins University
and Hospital)*

THE blood sera of human beings who have recovered from an attack of poliomyelitis and of monkeys (*Macacus rhesus*) which have recovered from the experimental disease are known to contain antibodies active against the virus,^{2,7,16} but the production of an artificial immune serum (hyper-immune) has not yet been accomplished. Favorable results have been obtained by the use of blood serum from human convalescents in the treatment of the experimental infection in monkeys^{17,18} and of the disease in human beings.^{15,19,20,21} However, these results have fallen short of the expectations which the demonstrated immunological properties of this serum would seem to warrant. The methods heretofore employed in the serum treatment of this disease have been chiefly intravenous and intraspinal injections, but a consideration of the modern conception of the circulation of the fluids of the central nervous system tissues raises the question as to whether

*Reprinted from Johns Hopkins Hospital Bulletin, Nov., 1923.

†Maintained by a special fund privately donated.

or not these methods of administration provide the optimal conditions for the therapeutic action of the serum. Since human immune serum can be obtained only in limited quantity and on account of the fact that it cannot be expected, by reason of its low potency, to yield the results which might be obtained with an hyper-immune serum, it is important that, pending the development of a more efficacious serum, every effort be made to obtain the maximal effect of this serum. Recent advances in the knowledge of the circulation of fluids of the central nervous system and the experimental production of alterations of these fluid currents suggest possibilities in this connection, and form the basis of the experiments to be recorded here.

MECHANISM OF LOCAL SPECIFIC THERAPY

Effective local specific therapy in disease of parasitic origin may be accomplished where it is possible to cause a suitable chemical or biologic agent to act on the parasites in a sufficient degree of concentration and for such a period of time as is necessary to accomplish their destruction without, of course, causing undue injury to the tissues of the host.^{32,37} It has been shown, for example, that the higher the concentration of the specific curative agent about the parasites in trypanosomal, meningococcal, and other infections, the greater is the assurance of their complete destruction. The availability of any remedial agent introduced into the body for this purpose is determined by its distribution there, and this in turn is dependent upon (a) the ability of a substance when introduced into one portion of the body to be carried by its physical or chemical affinities to other portions which harbor the micro-organisms, or (b) the ability to introduce the substance directly into the focus of the infection. There are excellent instances of the fortunate combination of circumstances which permit the utilization of each of these mechanisms for obtaining the de-

sired distribution of remedial agents. For example, tetanus antitoxin introduced into the subcutis for prophylactic purposes is carried to other points at which its action is desired in sufficient amount to bring about the neutralization of the toxin as it is liberated at the seat of the infection; whereas the fact that one is able to introduce antimeningococcic serum directly into the subarachnoid space makes possible a therapy of a degree of effectiveness which could not be brought to bear upon this disease if it were possible to introduce the serum into the circulation only, for example, and to rely upon its affinities for tissues to reach the lesions within the meninges.

In other instances the circumstances are not so fortunate. By reason of the fact that the parasites are so situated within the body that they do not lie within the paths of distribution of the particular therapeutic agent which are the result of its affinities for certain tissues, or if the parasites lie in a region of the body into which the curative agent may not be introduced directly, then either of the mechanisms referred to becomes ineffective. This is especially true of the tissues of the central nervous system. It is a striking fact that therapy in diseases in which the parasites exist within the central nervous system tissue is lamentably ineffective despite the fact that the chemicals or sera employed, as evidenced by tests against the microorganisms concerned, are suitable for their complete destruction. That this failure, at least in some instances, is due to the inability of the medication to reach the parasites is amply shown by the fact that lesions in more accessible portions of the body caused by the same organisms respond readily to such remedies, as, for example, in the case of syphilis of other tissues as compared with syphilis of the central nervous system.

The virus of poliomyelitis has not been found in the blood or cerebrospinal fluid at any stage of the disease in human

beings.^{6,36} In certain experiments on the route of infection, in which the monkey was used as a test animal, the virus was detected in the cerebrospinal fluid soon after its injection intravenously and in other experiments after its application to the nasal mucosa. However, after intravenous injection it tends to disappear from the blood stream so that after 120 hours it can no longer be detected there,³⁵ and although the virus was present in the cerebrospinal fluid on the third day after intracerebral inoculation,⁹ it was not demonstrable in it after the onset of symptoms of the experimental disease in the monkey.⁴ Thus, any appearance of the virus in the blood or in the cerebrospinal fluid is transitory and may be regarded as representing a stage in its passage from the exterior to the central nervous system tissue. Once localized in the central nervous system tissue, it does not again appear in the blood or cerebrospinal fluid during the course of the infection. Hence it would appear that the introduction of immune serum into either one of these fluids does not of itself insure contact of the immune serum with the invading virus.

INTRAVENOUS SERUM TREATMENT

Any substance in order to be delivered to the tissue cells from the circulation must pass through the walls of the capillaries. This endothelial wall exercises a high degree of specificity in regard to the substances which it allows to pass. This discriminatory power of the capillaries varies in different portions of the body in accordance with the needs of the particular tissues concerned, and in the case of the capillaries of the central nervous system, namely, the vessels of the brain and spinal cord and the specialized choroid plexus, it reaches a high degree of perfection.³³ These vessels are known to be capable of excluding most substances, including immunity principles, which may be present in the blood, from the tissue of the central nervous

system.³⁴ The cerebrospinal fluid is derived from the blood stream through the medium of these capillary systems and the character of this fluid itself is evidence of the precision of the apparatus which secretes it. These considerations indicate that immune serum introduced into the circulation would be entirely excluded from the tissue spaces of the central nervous system and this seems to be the case, provided the capillary walls which guard the entrance to these tissue spaces are intact. However, certain changes occur, or may be induced, which alter the permeability of these vessels and these should be taken into consideration if a proper understanding of the action of intravenous injection upon the central nervous system is to be arrived at.

Flexner and Amoss⁸ found that an irritation or even a slight alteration in the integrity of the meninges or choroid plexus, resulting from the subarchnoid injection of normal horse serum or other substances, permits the virus of poliomyelitis, when introduced into the circulation, to pass to the central nervous tissue and set up infection. Moreover, they demonstrated that immunity principles, although present in the blood as early as the sixth day of the disease,¹⁷ pass to the cerebrospinal fluid with difficulty,³ and suggest that the inflammatory condition present in the meninges might account for this passage of immunity principles from the blood to the cerebrospinal fluid and thus comprise a fortunate element in the pathological events of the disease. Flexner and Amoss have also shown that immunity principles, when injected into the blood stream, can be made to pass to the cerebrospinal fluid under conditions in which the meninges have been experimentally inflamed.³ Numerous observations of a similar nature have been recorded^{27,28,29,30} in respect to the passage of other substances from the circulation to the cerebrospinal fluid, and form the basis of the clinical application of an artificial disturbance of the relation between the blood stream and the cerebro-

spinal fluid spaces in the treatment of certain disease of the central nervous system.

The term "permeability of the meninges" has come into rather common use in reference to the passage of substances from the blood stream to the cerebrospinal fluid, but the exact manner in which such passage takes place is not yet known.

The fluid of the subarachnoid spaces is the combined product of the choroid plexus and the capillaries within the central nervous tissue itself.³¹ These structures, as well as the capillaries of the meninges and the posterior root ganglia,²² have been suggested as possible points of entrance into the central nervous tissue for substances coming from the circulation under the influence of a disturbance which brings about what is called an increased permeability of the meninges; and since fluid derived from each of these structures follows a different course within the central nervous system, it is to be expected that a substance delivered to the central nervous tissue from the circulation would depend for its distribution there upon whether it comes through one or another of these pathways. The criteria for affirming the passage from the circulation to the nervous tissue are the setting up of infection within the nervous system or the demonstration of the test substance in the cerebrospinal fluid, but in neither case may it be said with certainty through which gateway it has passed, and therefore the pathway which it has traversed within the central nervous system remains in doubt. While it is true in the case of infection with the virus of poliomyelitis that the process may be found within the nervous tissue itself, it does not follow that the infective agent was deposited there by the capillaries of that region, for indeed it has been shown that lesions are always present in the meninges⁵ even very early in the disease, and that intraspinal inoculation causes infection, although the virus disappears rapidly from the sub-

arachnoid space.^{5,36} Thus, by extension (multiplication of the virus) it reaches the tissue spaces within the brain and cord. In the case of the appearance of test substances in the cerebrospinal fluid, it is to be recalled that the fluid as obtained by lumbar puncture represents the combined product of the choroid plexus and the perivascular system; hence the presence of a substance in this fluid gives no indication as to which one of the pathways it has traversed. Nor can it be supposed that a substance dissolved in cerebrospinal fluid would enter the substance of the central nervous system in a manner similar to that which may take place in the case of a multiplying virus.

The fluid elaborated by the choroid plexus into the lateral ventricles of the brain flows through the foramen of Monro to the third ventricle, then to the fourth ventricle and through the foramina of Magendie and Luschka to the subarachnoid space, from which it is finally drained into the dural sinuses through arachnoid villi and to a certain extent along the sheaths of cranial and spinal nerves, finally to be taken into lymphatic channels. Thus it would appear that that portion of the cerebrospinal fluid coming from the choroid plexus is never brought into intimate contact with the central nervous tissue itself but remains without the boundaries of the nervous structure, namely, the pia mater and the ependymal lining of the ventricular system. It follows, then, that immune serum coming from the circulation along with this fluid would have little chance of being brought into contact with poliomyelitic lesions within central nervous tissue.

In like manner any fluid discharged from the capillaries of the meninges would pass immediately to the fluid in the subarachnoid space to be carried away along with the fluid from the choroid plexus.

There remains to be considered that portion of the cerebrospinal fluid which is elaborated by the capillaries within

the nervous tissue itself. This fluid passes through the walls of capillaries and circulates within the perivascular-perineuronal system, carrying nutritive substance to, and removing waste products from the cells, and finally discharging into the subarachnoid space and becoming a part of the common cerebrospinal fluid. It is this fluid alone which bathes the cells of the central nervous tissue, and only through it can it be expected that immune serum would reach the cells of the nervous structure from the circulation. In order for such a passage of immunity principles to take place it would be necessary that the integrity of this capillary system be altered. As has been pointed out, the exact mechanism of the changes which permit the passage of immune bodies from the circulation to the cerebrospinal fluid involves especially the blood vessels of the meninges and then the choroid plexus. Involvement of the capillaries within the nervous tissue has not been demonstrated in this connection, and from the location of these capillaries in reference to the meningeal surfaces such an assumption is hardly justified, so that it may be considered highly improbable that these capillary walls are the gateway through which substances pass.

With these considerations in mind the statement may be made that the intravenous administration of immune serum for the treatment of acute poliomyelitis is beset with the following difficulties:

(a) The great dilution of the serum in the circulation renders only a small proportion of it available for passage to the site of the lesions.

(b) The barrier between the circulating blood and the central nervous tissue, the capillaries of the central nervous tissue itself, choroid plexus and meningeal blood vessels would tend to reduce still further the proportion of serum which gains entrance to the central nervous tissue.

(c) Given the conditions under which immunity sub-

stances are permitted to enter the cerebrospinal fluid from the circulation, it is still, on account of the particular portion of the barrier which is broken down by the means at hand and the distribution within the central nervous system that which it permits, highly improbable that they are brought into contact with the main lesions of acute poliomyelitis. In this connection it should be stated that, although the vessels of the nervous tissue are involved in poliomyelitis, no increase in their permeability has been demonstrated.

INTRASPINAL SERUM TREATMENT

It has been shown by Weed³¹ that true solutions introduced into the subarachnoid space fail to enter the perivascular system of the central nervous tissues but join the current of the fluid contained in this space which is, as was pointed out in a previous paragraph, over the surfaces of the meninges and toward the exits from the central nervous system into dural sinuses. Clinically, the reaction of meningococcus lesions to immune serum introduced into the spinal subarachnoid space affords additional evidence of such a pathway. Lesions along the surface of the meninges (the typical meningitis) yield readily to serum administered in this way, whereas lesions within the nervous tissue, or in portions of the subarachnoid space, which are not in direct communication with that portion into which the serum has been introduced, do not respond to the same procedure.

Flexner and Amoss²⁴ in experiments upon the neutralization of the virus of poliomyelitis with immune serum found that by the introduction of an immune serum into the subarachnoid spaces, the virus is capable of being neutralized within the cerebrospinal fluid into which it is directly introduced, or to which it passes in transit from the blood to the nervous tissues. In the latter case neutralization prob-

ably is effected at successive stages in the process of transit of the virus from the blood to the nervous tissues. They considered it highly probable that the neutralization was accomplished before any quantity of the virus became attached to the nervous tissues themselves, earlier experiments by Flexner and Lewis²⁵ having shown that even when minute amounts of the virus were inoculated intracerebrally, neutralization was accomplished with very great difficulty by intraspinal injections of immune serum. In other words, when once attached to the central nervous system tissue, the virus is not neutralized by subarachnoid injection.

In connection with this failure of neutralization within the brain substance the experiments of Murphy and Sturm²⁶ upon the fate of tissue inoculated into the brain are perhaps of interest and emphasize the difference in reaction between the central nervous system tissue and its adjacent structures. They found that a transplantable mouse sarcoma grew readily when inoculated into the cerebrum of mice, provided the graft did not come in contact with the ventricle, in which case a cellular reaction occurred, similar to that about a subcutaneous heteroplastic graft. A bit of the animal's own spleen inoculated into the brain, along with the heteroplastic tumor tissue, prevented the growth of the foreign cells. Mice immunized to transplanted tumors were inoculated with grafts of mouse carcinoma, both subcutaneously and into the brain. Subcutaneous inoculation produced tumors in only 21 per cent of the animals, whereas the grafts into the brain produced tumors in 89 per cent. Control non-immunized mice inoculated with the same material developed brain tumors in 91.9 per cent and subcutaneous tumors in 82.2 per cent. Thus, mice highly resistant to subcutaneous transplants gave no evidence of this resistance when the tumor was inoculated into the nervous tissue of the brain.

There is now fairly complete evidence that the virus of poliomyelitis comes to be attached to a portion of the central nervous system which corresponds to that structural area into which immune bodies cannot be made to enter by either of the mechanisms of distribution referred to earlier in this paper. This seems to offer an explanation for at least a part of the lack of success with the serum treatment of this disease, and emphasizes the necessity of seeking other methods for obtaining a more adequate distribution of this serum within the central nervous system.

THE PASSAGE OF SUBSTANCES FROM THE SUBARACHNOID SPACE INTO THE PERIVASCULAR SYSTEM

Weed³¹ found that solutions of potassium ferrocyanide and to a certain extent carbon granules in suspension, introduced into the subarachnoid space, pass into the perivascular system of the brain when this organ is rendered anemic. From this he reasoned that there must be suction of fluid from the subarachnoid space to fill the perivascular spaces and make up for the loss of fluid in the brain resulting from anemia. Thus, the flow of fluid within the perivascular spaces which normally is toward the subarachnoid space was reversed. The method employed for the production of the anemia was tying the carotids or exsanguination. The effects produced were attributed to changes in the osmotic relationship between the blood stream and the cerebrospinal fluid. Later, Weed and McKibben²³ found that the simpler procedure of injecting strongly hypertonic solutions, such as a 30 per cent solution of sodium chloride into the circulation produced a marked reduction in the cerebrospinal fluid pressure. This finding indicated that this procedure would also serve to reverse the direction of flow within the perivascular system, as Weed and Hughson¹ have shown that the reduction in the cerebrospinal fluid pressure is due not to increased absorption of fluid into the

dural sinuses alone, but to a withdrawal of fluid from the tissue spaces into the capillaries. The addition of a foreign test solution to the subarachnoid fluid amply confirmed this point. When the cerebrospinal fluid pressure was falling rapidly or after it had become negative, following the intravenous injection of hypertonic salt solution, two or three cubic centimeters of a potassium ferrocyanide and iron ammonium citrate solution were allowed to flow into the subarachnoid space. By subsequent fixation of the tissues in acidified formalin this solution was precipitated as Prussian blue at the points which it had reached. The solution was found to have passed from the subarachnoid space along the perivascular spaces, reaching the interfibrous spaces in the white matter and the pericellular spaces in the grey matter.

These experiments, the general findings of which have been confirmed by numerous observers,^{10,11,12,13,14,38} indicate that the intravenous injection of hypertonic solutions of sodium chloride brings about new ratios between secretion and absorption of cerebrospinal fluid and causes a dislocation of a considerable quantity of the contents of the subarachnoid space into the perivascular-perineuronal system. They suggested the possibility of obtaining a more effective therapy with convalescent serum in poliomyelitis by placing it in the subarachnoid space and subsequently injecting hypertonic solutions into the circulation.

EXPERIMENTAL

The alterations in the fluid circulation of the central nervous system which form the basis of our therapeutic experiments have been made only on normal animals. On account of the particular conditions which exist in those structures which are chiefly concerned in the alterations spoken of in the presence of poliomyelitis infection, there was some question as to whether the same alterations would

be produced in the œdematous infiltrated tissues of this disease.

Exp. 1.—*Macacus rhesus* 27 was inoculated* intracerebrally with 2.5 c.c. of a suspension of glycerolated human spinal cord which contained the virus (Brow strain). On the ninth day after inoculation, the animal showed excitability, tremors, paralysis of the arms. On the following day the paralysis had increased in the arms and was beginning in both legs.

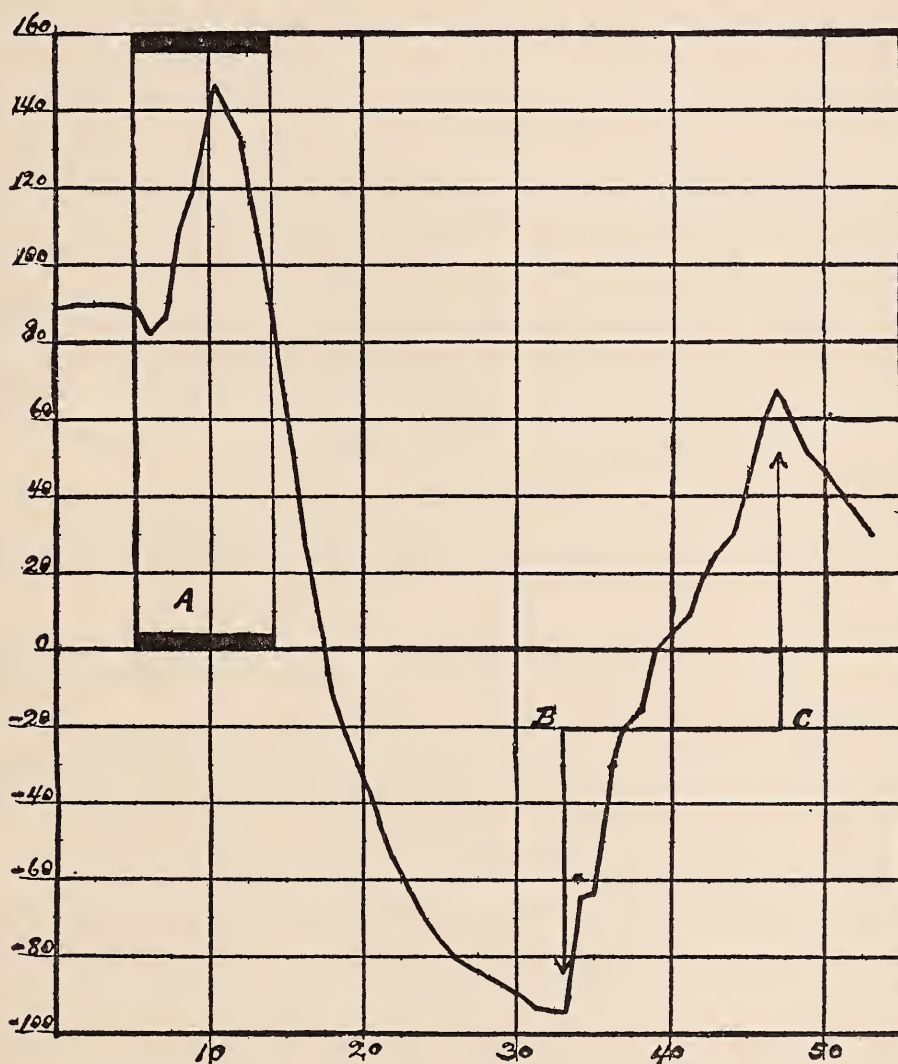


Chart 1. Experiment 1. Monkey 27 in acute stage of poliomyelitis. Weight 1320 grams. Ordinates represent cerebrospinal fluid pressure in millimeters of water; abscissae represent time in minutes. During blocked interval A, intravenous injection of 12 c.c. of 30 per cent solution of sodium chloride. During the interval from B to C, introduction of 3.5 c.c. of a solution of 0.5 per cent potassium ferrocyanide and 0.5 per cent iron-ammonium citrate.

*All inoculations and other operative procedures were carried out under ether anaesthesia.

Injection experiment.—Under ether anesthesia (which was maintained at a constant level by insufflation from a Woulfe bottle which did not require changing during the course of the experiment) a lumbar puncture was performed and the needle at once connected with a U-shaped manometer filled to zero with Ringer's solution. Cerebro-spinal fluid pressure was recorded at one minute intervals throughout the experiment. After 5 minutes, during which the pressure ranged from 84 to 90 mm. of Ringer's solution, 30 per cent sodium chloride was slowly injected intravenously. A total of 12 c.c. was given in 9 minutes. Within 4 minutes after the beginning of the intravenous injection the pressure rose to 146 mm. of Ringer's solution. It then fell rapidly to minus 94 mm. after 27 minutes from the beginning of the intravenous injection. At this point 3.5 c.c. of a solution of potassium ferrocyanide and iron ammonium citrate was introduced into the subarachnoid space through a burette attached to the manometer connection by means by a three-way cock. This solution was allowed to run in slowly during a period of 13 minutes. The addition of this amount of fluid caused the pressure to rise to plus 67 mm., after which it fell within the 6 minutes following to plus 30 mm., at which point the experiment was terminated. The tissue was fixed and the cyanide-citrate precipitated as Prussian blue by injection into the aorta of 10 per cent formalin containing 1 per cent hydrochloric acid. Microscopic examination of sections showed granules of dye distributed in the perivascular spaces. A full report of the distribution of the dye will be given in another communication.

Exp. 2. M. rhesus 23.

- December 6, 1921. Inoculated intracerebrally with 0.5 c.c. 5% suspension of glycerolated spinal cord of Monkey 19.
- December 17, 9 A. M. Apparent weakness of right hamstring.
8 P. M. Partial paralysis of extensors of right arm.
- December 18, 10 A. M. Paralysis of right arm and right leg. Tremors. Staccato cry. 3 P. M. 7 c.c. 30% sodium chloride solution intravenously (1.05 grams sodium chloride per kilogram).
- December 19, 9 A. M. Condition unchanged. 10 A. M. 6 c.c. 30% sodium chloride solution intravenously (.9 grams sodium chloride per kilogram). 5 P. M. Condition unchanged.
- December 20, 9:30 A. M. Paralysis stationary in right leg. Right arm "improving." 2:30 P. M. 6 c.c. 30% sodium chloride solution intravenously (.9 grams sodium chloride per kilogram).
- December 25, Slight Improvement.

The animal recovered with complete paralysis of the right leg and a slight involvement of right arm.

Exp. 3. M. rhesus 18, weight 2 kilograms.

- December 6, 1921. Inoculated intracerebrally, right side, with 0.5 of a 5% suspension of glycerolated cord of Monkey 19.
- December 13, 6 P. M. Excitable.
- December 14, 9 A. M. Excitable, head tremors, ataxia. Right facial weakness. 11 A. M. Right facial paralysis. 2 P. M. Paralysis of extensor muscles of both arms. Weakness of flexors of right arm. 4 P. M. 5 c.c. 30% sodium chloride solution intravenously (.75 gram sodium chloride per kilogram).
- December 15, 9 A. M. Paralysis of arms increased. 5:30 P. M. 6 c.c. 30% sodium chloride solution intravenously (.9 grams sodium chloride per kilogram).
- December 16, Paralysis extended in legs. 10 A. M. 6 c.c. 30% sodium chloride intravenously. 5 P. M. condition unchanged.
- December 17, 9 A. M. condition unchanged. 8 P. M. Paralysis has extended. 8:30 P. M. 2 c.c. convalescent serum intraspinaly.
- December 18, 9 A. M. Somewhat improved, general appearance better. 11:45 A. M. 4 c.c. 30% sodium chloride solution intravenously. 11:50 A. M. Respiration ceased.

Exp. 4. M. rhesus 5.

- December 18, 1922. 11:45 A. M. Inoculated intracerebrally with 2 c.c. of a 10% suspension of glycerolated poliomyelitic human spinal cord (Brow strain). A needle introduced into the lumbar subarachnoid space just before the intracerebral injection yielded no fluid until approximately 1 c.c. of the virus had been introduced into the cranial cavity, after which it began to drop slowly. The animal recovered in a few minutes from the ether anaesthesia and during seven days following was entirely normal in appearance.
- December 26, 9 A. M. Tremors of head. Ruffled coat, excitable. Paralysis of both arms, weakness of back and paralysis of adductors of right thigh. 12 M. Under ether anaesthesia. 20 c.c. of 30% solution of sodium chloride were injected intravenously at the rate of 1 c.c. per minute. Immediately after the beginning of the injection of sodium chloride solution, a needle was introduced into the lumbar subarachnoid space and connected to a manometer from which con-

valescent serum was allowed to flow into the subarachnoid space at a pressure which was not allowed to exceed 120 mm. of water. Fifteen minutes after the beginning of the injection of hypertonic salt solution the serum flowed quite rapidly into the subarachnoid space, this continuing for thirty minutes, at which time 10 c.c. had been used. The animal recovered from anæsthesia promptly and had a moderate chill. Within an hour, although still sluggish, was trying to sit up.

December 27,

9 A. M. No increase in paralysis. General appearance much better than before beginning of treatment. 12 M. Treatment was again undertaken, but after 12 c.c. of hypertonic salt solution had been given intravenously and 4 c.c. of serum intraspinally, respiration ceased.

Exp. 5. *M. rhesus* 29.

March 16, 1923.

Inoculated intracerebrally with 1 c.c. of a 5% suspension of glycerolated brain and cord of monkey 5.

March 30,

9 A. M. Excitable. Tremors. Weakness in both arms. 11 A. M. Paralysis of arms. Marked weakness in both legs. 2:50 P. M. Lumbar puncture was done and the needle connected with a burette-manometer. Cerebrospinal fluid pressure was recorded between 85 and 90 mm. water. 2 c.c. of human convalescent serum were allowed to flow into the subarachnoid space, which caused a rise in cerebrospinal fluid pressure to 130 mm. water. Occipito-atlantoid puncture was then done and the cerebrospinal fluid allowed to drip until its pressure had fallen to 85 mm. water. The fluid from the occipito-atlantoid puncture was at first clear and later showed an admixture of serum. Ten cubic centimeters of 30% solution of sodium chloride were then given intravenously at the rate of 1 c.c. per minute. This caused a rapid reduction of cerebrospinal fluid pressure. At intervals, as the pressure fell, small amounts of serum were allowed to run into the subarachnoid space so that the cerebrospinal fluid pressure was not raised above 100 mm. water.

This animal recovered from the ether anæsthesia promptly but was quite sluggish the rest of the day.

March 31,

Does not appear ill. Is alert and makes efforts to move about. No extension of paralysis.

April 1,

General appearance better. Improvement in general appearance in this animal was prompt, but there was some paralysis in all extremities which persisted.

Exp. 6. M. rhesus 31. Weight 1420 grams.

April 7, 1923. Inoculated intracerebrally with 1 c.c. of a 5% suspension of the glycerolated cord of Monkey No. 5.

April 16, 8 A. M. Excitable. Ruffled coat. Head tremors, slight ataxia.
4 P. M. Same. No paralysis.

April 19, 8:30 A. M. Paralysis of extensor muscles of both arms and of both legs, more marked in right. 11:30 A. M. Occipito-atlantoid puncture. 3 c.c. of convalescent human serum introduced into cerebellar cistern, which caused the cere-

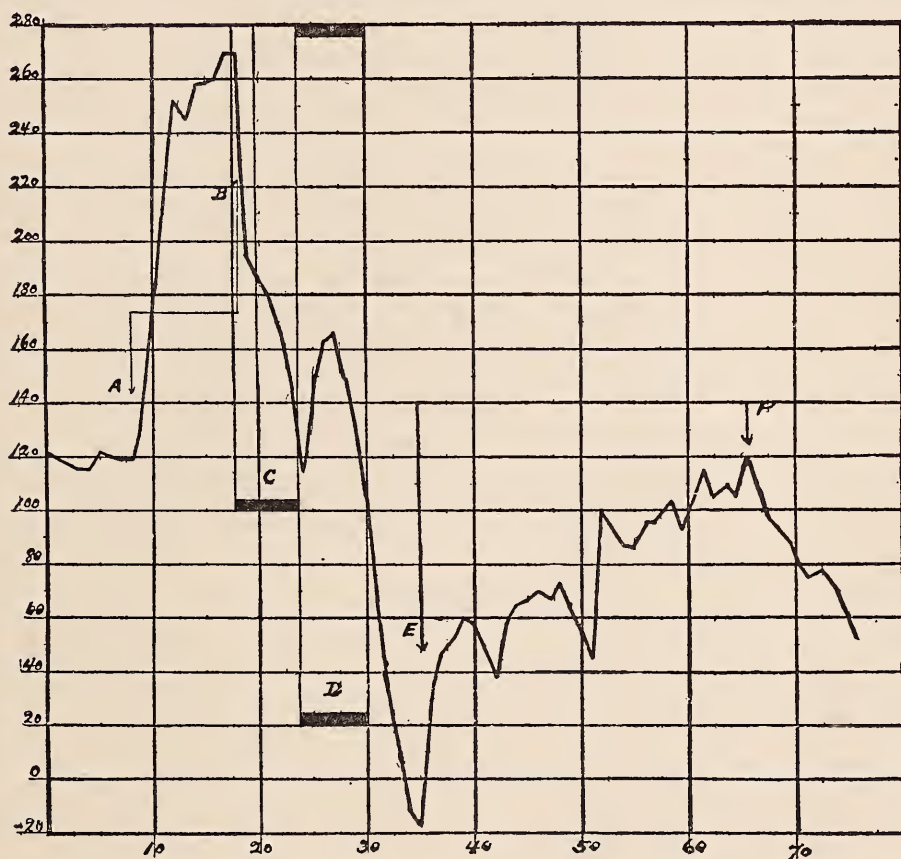


Chart 2. *Exp. 6. Monkey 31, in acute stage of poliomyelitis.* Ordinates represent cerebrospinal fluid pressure in millimeters of water; abscissae represent time in minutes. During the interval A to B 3 c.c. of convalescent human poliomyelitis serum were introduced into the cerebellar cistern. During the blocked interval C, cerebrospinal fluid was released from the subarachnoid space through lumbar puncture. During the blocked interval D, 13 c.c. of a 30% solution of sodium chloride were injected intravenously. During the interval E to F 7 c.c. of convalescent human poliomyelitic serum were introduced into the subarachnoid space.

M. rhesus No. 30, Control for No. 31.

brospinal fluid pressure to rise to 270 mm. water. This caused some respiratory embarrassment, which was relieved by lumbar puncture with a reduction of cerebrospinal fluid pressure to 115 mm. water. 13 c.c. of 30% sodium chloride solution intravenously at the rate of 2 c.c. per mm. Cerebrospinal fluid pressure rose to 165 mm. water and then fell rapidly to minus 16 mm. water. 7 c.c. convalescent serum slowly introduced into the cerebellar cistern. 4 P. M. Somewhat slow but gets about cage. 12 c.c. convalescent serum intravenously.

- April 18, Sitting up. Appears bright. No extension in paralysis.
- April 19, Improved. Gets about with ease.
- May 17, Recovery with partial paralysis of right leg.
- April 7, 1923. 3:15 P. M. Inoculated intracerebrally right side with 1 c.c. 5% suspension of glycerolated cord of Monkey No. 5.
- April 17, 8 A. M. Excitable, marked head tremors, moderate ataxia. 9 P. M. Excitable, marked head tremors, more ataxic.
- April 18, 9 A. M. Excitable, tremors, more ataxic; misses hold.
- April 19, 9 A. M. On floor. Flaccid paralysis of extensors of arms. Flexors good. Some weakness of legs.
- April 20, Both legs flaccid.
- April 21, Down, barely able to move arms. Appears ill; head tremors.
- April 22, Paralysis same. Head tremors. Ill.
- April 23, Paralysis same. Head tremors. Ill.
- April 24, Paralysis increased. Almost prostrate. Head tremors.
- April 25, Some edema under eyes. Some tremors.
- April 27, Same.
- April 28, Same.

Gradual subsidence of acute symptoms. Unable to use extremities except hands. Gets about cage by pulling with hands and twisting body. Remained in same condition until May 17.

- May 17, Sits up. Uses both arms and one leg fairly well.

SUMMARY AND DISCUSSION

The experimental findings presented indicate that the intravenous injection of strongly hypertonic solutions of sodium chloride causes marked alterations in the circulation of fluid within the central nervous system of monkeys in the acute stage of poliomyelitis (Exp. 1). These alterations are apparently similar to those reported by other observers in normal animals, namely, reduction of cerebrospinal fluid pressure, reduction in the volume of the brain and spinal cord (removal of fluid) and a reversal of the current of fluid within the perivascular spaces.

Reduction in the volume of the brain and spinal cord suggested the use of hypertonic intravenous injections as a means of reducing to some extent the edema of the central nervous system tissue in acute poliomyelitis. However, it is realized that factors other than osmosis are involved in edema. The course of the disease in one monkey treated in this manner (Exp. 2) as compared with the control animal in this series (Monkey 30), inoculated with the same strain of virus, is indicative of a beneficial effect of hypertonic solutions on the course of the disease.

Experiments 3 and 4 were designed to make use of the reduction in volume of the brain and spinal cord for lessening the edema as suggested by the previous experiment and at the same time to replace the contents of the subarachnoid space with human convalescent serum which would be drawn into the perivascular spaces, under the influence of the hypertonic solution, and thus bring about a distribution of the serum, which does not take place after administration by other methods. In both these animals there was evidence of improvement following treatment, but unfortunately attempts to repeat the injection of relatively small amounts of hypertonic sodium chloride solution caused death of the animals.

Experiment 5 was carried out in the same manner as

Experiments 3 and 4 except that only one injection was given. As shown on the protocol, this monkey had marked involvement when treated, but his condition on the following day was strikingly better. Recovery, although accompanied by a considerable degree of paralysis, was prompt as compared to the slow recovery of the control monkey 30.

Experiment 6 was a repetition of Experiment 5 with the addition of an intravenous injection of serum given 3 hours after the injection of hypertonic salt solution; in order that antibodies would be present in the blood when the flow of fluid from the circulation to the central nervous system fluid spaces was re-established. This animal showed an almost immediate improvement. There was no extension of paralysis after the one treatment, and recovery was rapid with only a partial paralysis of the right leg. As compared with the usual course of the disease in monkeys the outcome of this experiment is regarded as very favorable.

CONCLUSIONS

This small series of experiments indicate that the use of intravenous hypertonic solutions in conjunction with intraspinal convalescent serum has a favorable influence on the course of experimental poliomyelitis. This effect is augmented by the intravenous injection of convalescent serum at a time corresponding to the compensatory increase in passage of fluid from the circulation to the central nervous system tissue.

The optimal conditions in respect to the administration of hypertonic solutions remain to be worked out.

Daily repetition of hypertonic sodium chloride solution is associated with danger of respiratory failure.

REFERENCES

- ¹Weed, L. H., and Hughson, W.: *Am. Jour. Physiol.*, 1921, LXIII, 1.
- ²Levaditi and Landsteiner: *Compt. rend. Soc. biol.*, 1910, LXVIII, 311.
- Romer, P. H., and Joseph, K.: *Münch. med. Wnschr.*, 1910, LVII, 568.

- ³Flexner, S., and Amoss, H. L.: Jour. Exper. Med., 1917, XXV, 499.
- ⁴Flexner, S., and Clark, P. F.: Jour. Amer. Med. Assn., 1911, 57.
- ⁵Draper, G., and Peabody, F. W.: Am. Jour. Dis. Child., 1912, III, 153.
- ⁶Clark, P. F., Fraser, F. R., and Amoss, H. L.: Jour. Exper. Med., 1914, XIX, 223.
- ⁷Anderson, J. F., and Frost, W. H.: Jour. Amer. Med. Assn., 1911, 54: 663.
- ⁸Flexner and Amoss: Jour. Exper. Med., 1917, XXV, 525.
- ⁹Flexner and Lewis: Jour. Amer. Med. Assn., 1910, 54: 14.
- ¹⁰Cushing and Foley: Proc. Soc. Exper. Biol. and Med., 1920, XVII, 217.
- ¹¹Sachs and Belcher: Jour. Amer. Med. Assn., 1920, 75: 667.
- ¹²Foley and Putnam: Am. Jour. Physiol., LIII, 464.
- ¹³Ebaugh and Stevenson: Johns Hopkins Hosp. Bull., 1920, XXXI, 440.
- ¹⁴Sachs and Malone: Am. Jour. Physiol., 1921, LV, 277.
- ¹⁵Amoss, H. L., and Chesney, A. M.: Jour. Exper. Med., 1917, XXV, 581.
- ¹⁶Flexner, S., and Amoss, H. L.: Jour. Exper. Med., 1917, XXV, 499.
- ¹⁷Flexner, S., and Lewis, P. A.: Jour. Amer. Med. Assn., 1910, 54: 1780; 55: 662.
- ¹⁸Netter, A., Gendron, A., and Touraine: Compt. rend. Soc. biol., 1911, LXX, 625.
- Netter, A.: Bull., Acad. de méd., 1915, LXXIV, 403.
- ¹⁹Netter, A., and Salanier, M.: Bull., et mém. Soc. méd. d. hôp: de Par., 1916, XL, 299.
- ²⁰Weekly Bull. Dept. of Health, City of New York, 1916, N. S., V., 345.
- ²¹Fisher, L.: Med. Record, 1917, XCI, 52.
- ²²Flexner, S., Clark, P. F., and Amoss, H. L., Jour. Exper. Med., 1914, XIX, 205.
- ²³Weed, L. H., and McKibben, R. S.: Am. Jour. Physiol., 1919, XLVIII, 531.
- ²⁴Flexner and Amoss: Jour. Exper. Med., 1914, XX, 249.
- ²⁵Flexner, S., and Lewis, P. A.: Jour. Amer. Med. Assn., 1910, 54: 1780; 1910, 55: 662.
- ²⁶Murphy, J. B., and Sturm, E.: Jour. Amer. Med. Assn., 1922, 79: 1159.
- ²⁷Osborne, E. D.: Jour. Amer. Med. Assn., 76: 1384-1387, (May 21) 1921.
- ²⁸Mehrtens, H. G.: Calif. State Jour. Med., 1918, XVI, 306.
- ²⁹Wegeforth, P., and Latham, G.: Amer. Jour. Med. Sc., 1919, CLVIII, 183.
- ³⁰Mehrtens, H. G., and MacArthur, C. G.: Arch. Neurol. and Psychiat., 1919, II, 369.
- ³¹Weed, L. H.: Jour. Med. Res., 1914, XXXI (new series, XXVI, 93).
- ³²Flexner, S.: Jour. Exper. Med., 1907, LX, 168.
- ³³Levy and Gissler: Münch. med. Wehnschr., 1897, XLIV, 1435.
- ³⁴Strauss and Wolff: Fortschr. d. Med., 1902, XX, 1, 209.
- ³⁵Flexner, S., and Amoss, H. L.: Jour. Exper. Med., 1914, XX, 249.
- ³⁶Clark, P. F., and Amoss, H. L.: Jour. Exper. Med., 1914, XIX, 217.
- ³⁷Blackfan, K. D.: Medicine, 1922, 1, 139.
- ³⁸Weed, L. H.: Amer. Jour. Anat., 1923, XXXI, 191.





University of
Connecticut
Libraries



39153029897909

